The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 129, No. 18 — CHICAGO, ILLINOIS — DECEMBER 29, 1945

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PAROXYSMAL ABDOMINAL PAIN

A FORM OF FOCAL SYMPTOMATIC EPILEPSY: II

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One of the commonest complaints attending man from the cradle to the grave is abdominal pain. To enumerate the array of causes for such pain is not the province of this paper, but, in general, they originate not only from intrinsic disorders of the abdominal viscera but also from lesions of the thoracic viscera, spinal cord and peripheral nerves, and the more remote brain. It is the very multiplicity of these causes which often makes differential diagnosis difficult and elusive.

There is a small group, sufficiently large however and inadequately recognized to warrant further emphasis, in which the abdominal pain may have been present over varying periods of time without a demonstrable organic or psychogenic cause having been unearthed by the usual methods of study. What heretofore may have been considered unexplained abdominal pain obviously must have an explanation, and it is suggested that where this variety of pain exists study be directed to the possible existence of cerebral disease or dysfunction.

Exact localization of cerebral representation of gastrointestinal activity and abdominal pain is still not fully determined, but most data point to cortical mediation and possibly diencephalic influence. Fulton [1] has reviewed the early experimental work, beginning with Bochefontaine (1876), Hlasko (1887) and Bechterew (1911), indicating the evidence of cortical influence on gastrointestinal motility. Recent studies by Fulton and his co-workers,[2] Spiegel, Weston and Oppenheimer [3] and Penfield and Erickson [4] have confirmed and expanded the evidence of gastrointestinal representation in the cortex (areas 6, 5, 3 of Brodmann). Penfield and Gage [5] have shown that abdominal pain may occur as an epileptic aura or an equivalent, and they describe pain arising in the lower right quadrant of the abdomen followed by a convulsive seizure in a patient whose exposed brain was stimulated electrically in area 5a. In calling attention to the important subject of brain trauma and its relationship to disturbed function and production of lesions of the gastrointestinal tract, Cushing [6] showed the probable role played by the diencephalon in this respect. Watts and Fulton [7] also have demonstrated the effects of diencephalic lesions on the gastrointestinal tract.

From the clinical approach abdominal pain has been postulated as being a form of epileptic variant by Wilson [8] and demonstrated in pure form by Moore.[9] Klingman, Langford, Greeley and Hoefer [10] have reported the cases of 12 children having unexplained attacks of paroxysmal abdominal pain in whom, by the history of associated manifestations of the convulsive state and the electroencephalographic findings of cerebral dysrhythmias, they concluded that the type of pain was an expression of an epileptic disturbance. Lambert [11] too has suggested that abdominal pain in children may represent a form of the convulsive state secondary to altered cortical function, as shown by electroencephalographic tracings in which the cerebral dysrhythmias were of the petit mal and psychomotor equivalent types.

Fits of varying forms, including abdominal pain, may result from a host of conditions producing either structural or physiologic changes in the brain. These causes have been amply enumerated and discussed by Cobb.[12] In the consideration of abdominal pain as a symptom of cerebral disease, Wechsler [13] stated "I would call attention to the occurrence of abdominal pain, at times simulating appendicitis, renal colic, gallbladder disease and gastric or duodenal ulcer, in tumors of the brain. I have seen patients treated for a long time and even operated upon for abdominal syndromes which were the harbingers of brain tumors."

The following case reports constitute a group of individuals in whom abdominal pain was either the only symptom or an outstanding one, present and unrecognized over a considerable period of time, and in whom thorough history taking, detailed neurologic

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Paper published under the auspices of the Section on Nervous and Mental Diseases.

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examination and utilization of diagnostic aids including electroencephalography led to the consideration of the pain as a manifestation of focal symptomatic epilepsy.

CASE 1.[14]—P. P., a man aged 32, was referred by Dr. Marcel Sussman, June 4, 1941, with the chief complaint of periodic attacks of cramplike pain involving the entire abdomen. The pain often started in the lower right quadrant and then involved the abdomen throughout. At times the pain became so severe as to produce prostration and loss of weight. During the attack he had a "rumbling" sensation in the abdomen associated with considerable eructation. The seizures were not accompanied by headache or visual disturbances. He had never had diarrhea during a spell. Following the attack he was exhausted and felt "achy all over." The first attack occurred at the age of 9 months following the injection of diphtheria antitoxin. The attacks of abdominal pain came on at irregular intervals during his early life but during recent years have been very frequent, occurring about every seven or eight days, and during the week preceding the examination there were four seizures. At the age of 8 an appendectomy was performed during one of the attacks and a normal appendix was removed. He had been studied extensively and various diagnoses had been made, including hysteria, abdominal angioneurotic edema, abdominal migraine, psychogenic functional gastrointestinal disorder, hepatobiliary disease, enlarged colon, Dietl's crisis and diaphragmatic hernia. He received various forms of medication, including ergotamine tartrate, without obtaining relief. Both parents are living and there is no history of a similar condition existing in the immediate forebears. The neurologic examination showed no abnormal findings. Laboratory studies showed essentially normal findings, and previous x-ray studies of the gastrointestinal tract, urinary tract and gallbladder were negative. The blood pressure was 102 systolic, 68 diastolic, pulse rate 76, weight 139½ pounds (63 Kg.). Electroencephalography [9] performed by Dr. Joseph Hughes showed large, low voltage, abnormal waves seen particularly in the right and left frontal regions. They were also present in the parietal and occipital lobes. This abnormal activity was increased with hyperventilation. The patient was placed on anticonvulsant therapy consisting of diphenylhydantoin sodium (dilantin sodium) 1½ grains (0.1 Gm.) twice daily and a bromide mixture 1 fluidrachm (4 cc.) four times daily. He remained attack free for seven and a half months, when medication was withdrawn, resulting in a return of seizures. On resumption of medication he was free from attacks until July 16, 1942, at which time a placebo was substituted in the diphenylhydantoin sodium capsules (the bromide mixture having been discontinued for some time) and again the attacks returned within fifteen days. The patient was terribly alarmed, believing the diphenylhydantoin was no longer effective, but when told about the experiment he was gratifyingly reassured. During the time he was attack free his weight rose from 139½ to 163 pounds (from 60 to 74 Kg.). He has been taking diphenylhydantoin sodium 1½ grains twice daily regularly and to the present date has been entirely attack free.

This patient had previously been treated for numerous conditions, among which was so-called abdominal migraine. He failed to respond to ergotamine tartrate. The periodicity of the attacks, their paroxysmal, abrupt onset, and their relatively short duration made it appear that the symptom was an abdominal epileptic variant. As indicated in the full case report,[9] it was believed that the injection of diphtheria antitoxin had produced an angioneurotic edema of the brain with subsequent changes in the cerebral cortex which predisposed to abnormal cerebral discharges. These discharges were seen to occur predominantly in the frontal lobes, as indicated by electroencephalography. The intense abdominal pain may be essentially intrinsic in the intestine, secondary to intense visceral contractions brought about by the abnormal cerebral discharges in the frontal and/or the parietal cortex.

CASE 2.—A. S., a man aged 43, was admitted to the Jewish Hospital to the service of Dr. Harold Goldburgh on May 1, 1944 with the admission diagnosis of enterocolitis. For six months prior to his admission he had been suffering from frequent attacks of severe abdominal pain associated with diarrhea, cold sweats, weakness and occasional loss of consciousness. At first the attacks occurred every eight to ten days and then increased in frequency, so that during the month prior to admission they occurred every day at least once and often several times daily. The past medical history revealed that at the age of 10 he had a head injury with a period of unconsciousness, the exact duration of which is not known. At the age of 12 he developed spells consisting of a "peculiar sensation" in the abdomen followed by a "prickly" feeling over the entire body, followed immediately thereafter by a cramplike pressure in the abdomen associated with "rumbling" and an awareness of the immediacy of a bowel movement. This was followed by a watery evacuation. This attack usually was accompanied with profuse sweating. In addition to these attacks he had spells consisting of blanching of the face, unconsciousness and falling to the floor, followed by "twitchings" (convulsive movements?) of the arms and legs and at times frothing at the mouth. These seizures occurred at intervals of one to three months and continued up to the present disturbance, which began six months before his admission to the hospital. The past medical history revealed the usual childhood diseases. In 1939 he contracted undulant fever, for which he received fever therapy. There was nothing in the remainder of the history to indicate any emotional disturbances. The patient is married. Both parents are living and well. Three brothers and one sister are living and well; a fourth brother is a mongoloid idiot. There is no history of epilepsy in the immediate or collateral members of the family. The blood pressure was systolic 145, diastolic 90. Laboratory studies revealed blood calcium, urea, sedimentation rate, Wassermann reaction, urinalysis, gastric analysis and the feces all normal. The sugar tolerance test was normal. The leukocyte count showed a low normal of 5,000 cells with 6 per cent eosinophils. Examination of the chest and abdomen was negative. X-ray studies of the entire gastrointestinal tract were reported as indicating the presence of enterocolitis and duodenitis. X-ray examination of the skull showed no abnormalities. The spinal fluid examination was negative. The neurologic examination showed the cranial nerves to be normal; sensation was preserved throughout the body in all modalities and was equal on the two sides; the tendon reflexes in the upper limbs were extremely prompt and there was a distinct preponderance on the left side; the knee jerks and ankle jerks were overactive and again there was increased activity on the left as compared with the right. Pathologic reflexes could not be brought out. Electroencephalographic studies performed by Dr. Donald Scott showed "that the alpha rhythm of 9.4 per second was equal and very persistent on both sides of the head. The general level of activity was low normal, while the pattern showed no irregularities whatever. A satisfactory hyperventilation resulted in no change in the record. In the absence of any abnormality, the record was not suggestive of the nature or origin of the patient's complaint." It was suggested that the patient be given diphenylhydantoin sodium 1½ grains twice daily. The attacks ceased until June 1944, when he stopped taking medication. Within two weeks he developed attacks of abdominal pain, which occurred almost daily. During October 1944, at a time when he was not taking diphenylhydantoin sodium, the attacks occurred several times daily. He finally resumed medication on Dec. 12, 1944, taking ¾ grain (0.05 Gm.) of diphenylhydantoin sodium four times a day. There was immediate relief, and during a recent interview he stated that he has been feeling better than at any other time in his life.

14. This case is reported in full by Moore.[9]

The spells from which this patient has suffered over a period of thirty-one years usually occurred with explosive suddenness and often were preceded by a "peculiar" sensation which the patient had difficulty in describing adequately. This sensation is similar to that experienced in the epileptic aura. During the six month period preceding admission to the hospital he had predominantly abdominal pain as the main feature of his spells. These not infrequently terminated with unconsciousness, although not invariably so. There is a history of a head injury at the age of 10 and the onset of seizures at 12. Although the electroencephalogram failed to show abnormal cortical discharges, the immediate response to treatment with diphenylhydantoin sodium and the subsequent return of symptoms on cessation of treatment, and again the relief of the paroxysmal abdominal pain by resumption of anticonvulsant medication, would indicate the nature of the complaint being of an epileptic order. Gibbs and his associates [15] in their analysis of 175 cases of post-traumatic epilepsy have shown that the electroencephalographic evidence of focal cerebral discharges occur in 51 per cent and generalized discharges in 41 per cent and that normal electroencephalograms occur in 8 per cent. They also state, "however, it must be recognized that a normal electroencephalogram does not exclude the possibility that a patient has brain damage or post-traumatic epilepsy."

CASE 3.—A. D., a girl aged 3 years, was admitted to the Jewish Hospital to the service of Dr. Samuel Goldberg, May 24, 1944 with the admission diagnosis of rickets. She was unable to talk, obeyed commands indifferently and had no control of bowel or bladder function. For many months she had periodic, abrupt attacks of crying, screaming and thrashing about, coming on without apparent cause at irregular intervals during the day. If the child was spanked, she seemed to go into a stupor and became rigid. She had been delivered normally at full term. At the age of 5 months she developed attacks of abdominal pain, which caused her to double up and cry out. The attacks were of short duration and occurred both diurnally and nocturnally. Following the individual seizures of abdominal pain the patient urinated and then slept three to four hours. Roentgen studies of the entire gastrointestinal tract showed nothing abnormal. From 6 to 11 months of age there was a period of relative quiescence with only occasional attacks of abdominal pain. In 1942 studies revealed an enlarged thymus gland and low blood calcium. A diagnosis of intestinal tetany was made and the patient was treated by irradiation of the thymus gland and the administration of calcium salts. Although less frequent, the spells of abdominal pain persisted until July 1943, since which time the abdominal pain has ceased.

The child walked at 17 months but failed to develop normally as regards talking and behavior. Both parents are living and well. There is no history of rheumatic fever, syphilis, tuberculosis, diabetes, epilepsy or mental disease in the family. Ophthalmoscopy before admission to the hospital revealed bilateral optic atrophy.

The neurologic examination showed that the child was capable of walking but presented pronounced mental deficiency. Both fundi showed atrophic disks. There was a definite defect in hearing. Over the skin of the nose and maxillary region there were small elevations of the skin in a butterfly configuration characteristic of the nevus multiplex of Bourneville (sebaceum adenoma of Pringle). Complete laboratory studies, as regards blood chemistry, blood count, blood Wassermann, blood calcium and phosphorus and urinalysis were negative. X-ray examination of the skull revealed no abnormalities. The triad of mental deficiency, nevus multiplex of Bourneville and epileptoid manifestations first shown by abdominal pain and then by bouts of uncontrolled temper tantrums of a convulsive state pattern led to the diagnosis of tuberous sclerosis.

The diagnosis of tuberous sclerosis was made at the last admission to the hospital on the basis of the finding of the characteristic lesion over the skin of the face, together with mental deficiency and the interpretation of the abdominal pain and behavior disturbances as a convulsive state manifestation. The attacks of abdominal pain, which were originally diagnosed intestinal tetany, came on abruptly, were episodic and were of short duration, followed by exhaustion and deep sleep. This description fits into the seizure pattern of epilepsy. Another interesting phase of the history was the stupor and rigidity following on the child being spanked, which might well be the so-called reflex epilepsy.[16] Although pneumoencephalography was not performed because of refusal by the parents, one can visualize the characteristic morbid changes in the brain being well scattered and involving the premotor cortex in area 6 and the parietal-sensory cortex in areas 3 and 5, which presumably influence intestinal activity (2, 3). The substitution of the bouts of abdominal pain by abrupt attacks of abnormal behavior occurring without apparent cause is probably also an exhibition of the convulsive state.[17]

CASE 4.—N. T., a boy aged 11 years, was referred by Dr. Ehrich Urbach, Dec. 11, 1944 with the history of attacks of unconsciousness, three of which had taken place during the past four years, numerous interval seizures of abdominal pain, nausea, occasional vomiting and diarrhea. The first episode occurred four years before, at which time he was unconscious and had convulsive movements, followed by a period of confusion. After a free interval of one month he had a second seizure. During July 1944 he experienced an attack of severe abdominal pain, following which he vomited and had diarrhea, the entire spell lasting about ten minutes. Prior to this he had had periods, lasting several months, of intermittent seizures consisting of waves of abdominal pain which came on about twice weekly with free intervals of six months to one year. These bouts of abdominal pain usually lasted from two to three minutes and occurred at intervals of two to ten minutes apart, the total "wave" lasting approximately one hour. These waves of abdominal pain occurred either in the morning or at night and were not accompanied with diarrhea or associated with unconsciousness. During the past year he had had frightful nightmares and talked in his sleep. His most recent attack, taking place when he was in bed, occurred two weeks before the present examination. He developed a headache, was nauseated, had a desire to defecate, and developed pain in the lower abdomen. The pain in the abdomen persisted and he experienced a feeling of suffocation. While in bed he had a continuous desire to move his bowels. After a period of several minutes he lost consciousness. Later in the evening, when he was found by his parents, he seemed confused. Following these bouts of abdominal pain there was always a short aftermath of slight confusion.

Five years prior to examination he sustained a severe head injury, having struck his head against a tree while sledding down an incline. He was unconscious for twenty-four hours. His previous history as regards delivery was normal, and he had the usual childhood diseases such as measles, mumps and chickenpox. The child was somewhat pudgy. The neurologic examination revealed that he was alert, and there was no disturbance in his mental sphere. The cranial nerves showed no abnormalities. There were no disturbances in motor dynamics.

15. Gibbs, F. A.; Wegner, W. R., and Gibbs, Erna L.: The Electroencephalogram in Post-Traumatic Epilepsy, Am. J. Psychiat. **100**: 738, 1944.

16. Wilson, S. A. K.: Neurology, edited by A. N. Bruce, Baltimore, Williams & Wilkins Company, 1940, p. 1503.

17. Moore, M. T.: Aberrant Forms of Epilepsy: Their Disguise in Somatosensory, Psychic and Unusual Motor Displays, Pennsylvania M. J. **48**: 569, 1945.

Coordination, sensory endowments and tendon reflex activity were normal throughout. Examination of the chest and abdomen showed no morbid changes of the viscera. The blood pressure was systolic 98, diastolic 56; the pulse rate was 86, the weight 95 pounds (43 Kg.).

The patient developed attacks of abortive grand mal epilepsy one year following a severe head injury; these fits did not have a stereotyped or well defined pattern. The attacks were characterized mainly by unconsciousness, questionable clonic and tonic phase, postictal confusion, and on two occasions loss of bowel and bladder control. In addition to this there were episodes of abdominal pain which occurred at irregular intervals and which came on paroxysmally in waves. These attacks of abdominal pain, followed occasionally by a short period of confusion, might well be the result of abnormal discharges in the frontal or parietal lobes. It was felt expedient, therefore, to have electroencephalographic studies done as well as x-rays of the skull. The latter were negative. The former were interpreted by Dr. Donald Scott to be as follows (fig. 1): The alpha rhythm of 9 per second was equally persistent on the two sides of the head. The general level of activity was considerably increased above normal limits, owing largely to the presence of slow waves, which could be seen on all leads. These occurred both singly and in groups, and, while they gave no indication of a focal origin, they were somewhat more persistent in the frontal area than elsewhere. It was interesting that this type of activity was almost wholly abolished when the patient opened his eyes. In frequent instances this slow wave activity was organized into typical groups of spike and slow wave forms, which were bilaterally symmetrical. No hyperventilation was attempted. Impression: The decidedly abnormal activity which was seen in this record was strongly suggestive of the type of disturbance usually seen in cases of idiopathic epilepsy. In view of the history of trauma, particular care was taken to find a focal origin, but, while some groups of slow waves reversed on the left, an equal number reversed on the right and even more frequently the activity was bilaterally symmetrical. Also it has been our experience that, unless the damage is very severe, focal activity will not be seen from all areas of the cortex. In conclusion, the record most closely approximated the pattern seen in idiopathic epilepsy.

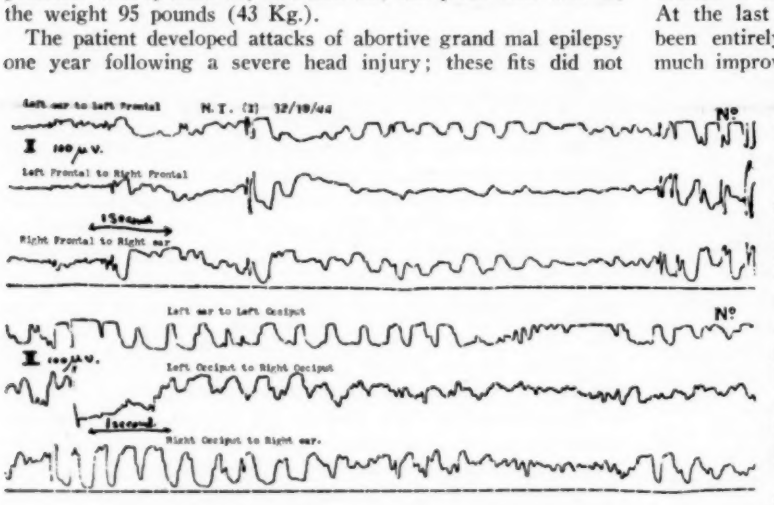

Fig. 1.—First electroencephalographic tracing in case 4.

It will be seen from the description of the electroencephalographic findings that the slow waves were found mainly in the frontal lobes, although there was a diffuse scattering of abnormal discharges throughout the brain. It was concluded, therefore, that the patient had a form of post-traumatic epilepsy manifesting itself in two varieties: one, an abortive form of grand mal (three seizures in four years), two, a frequent, interval variety assuming the characteristics of abdominal epilepsy.

On Dec. 26, 1944 the patient was directed to take 1½ grains of diphenylhydantoin sodium twice daily and calcium-bromine-galactogluconate (calcibronat-Sandoz) ½ drachm (2 Gm.) dissolved in water three times daily. Following this form of medication there was an almost immediate cessation of the attacks of abdominal pain, and no grand mal seizures occurred. At the last examination on March 13, 1945 the patient had been entirely free from abdominal seizures and had shown much improvement in his mental acuity.

Electroencephalographic examination with the patient receiving full medication revealed the following findings (fig. 2): April 5, 1945. The alpha ryhthm of 10 per second was equally persistent on the two sides of the head. The general level of activity was normal for a child of this age. The pattern closely resembled that which was seen before and consisted of a normal alpha rhythm, superimposed on which were numerous groups of slow waves, both with and without spikes. These could be seen on all leads but were most prominent in the frontal and occipital areas. They were usually bilaterally symmetrical, but not infrequently reversed on the left side. A satisfactory hyperventilation resulted in the appearance of two prominent groups of these waves in two minutes. Impression: The evidence pointed to a diffuse disturbance of cortical activity but it was not yet clear whether this was more properly considered as idiopathic epilepsy or some other form of diffuse damage. The fact that the slow waves were usually in groups and often were bilaterally symmetrical, as well as most pronounced in the frontal area, pointed to the first possibility, while the single spike and slow waves, which reversed on the left, looked more like scarring. Taken as a whole, the record showed some 30 per cent less abnormal activity than the earlier study.

This patient had sustained a severe head injury five years before first being seen by the examiner. One year thereafter he developed an overt loss of consciousness with convulsive movements, the exact nature of which were not adequately described by his parents. This seizure was followed by attacks of abdominal pain which came on abruptly, lasted from two to three minutes and which appeared at intervals in a "wave-like manner," the entire cycle lasting up to one hour. The attacks of abdominal pain at times were accompanied by nausea and diarrhea and occasionally with vomiting. The electroencephalographic findings of abnormal cerebral discharges, akin to those seen in idiopathic epilepsy, predominantly in the frontal lobes, would indicate that the seizures of abdominal pain represent an epileptic variant. Moreover, the immediate and continued response to anticonvulsant medication would again point to the relationship of the abdominal pain to cortical dysfunction. In addition,

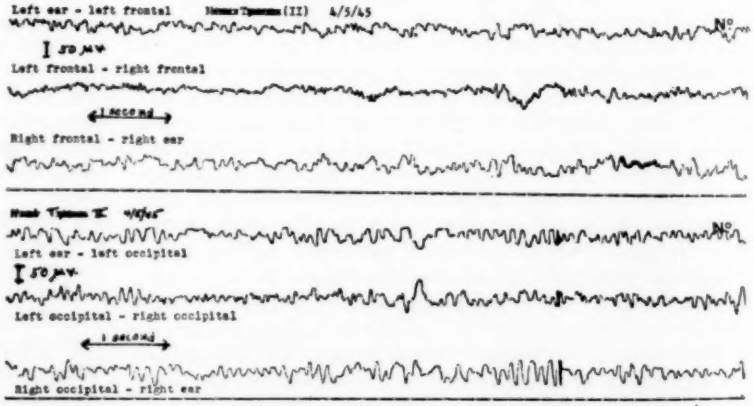

Fig. 2.—Second electroencephalographic tracing in case 4.

the electroencephalographic findings with the patient under medication (fig. 2), showing the 30 per cent damping out of the previously existing abnormal discharges, indicates the action of the anticonvulsant drugs [18] and the tendency to permit the focal nature of the discharges to come to the fore. It will be seen in this case that the dominant form of attack was that of paroxysmal abdominal pain occurring in a seizure pattern corresponding to that found in typical epilepsy. Only three attacks of abortive grand mal occurred during the five year period dating from the head injury. The abdominal pain was by far the more annoying and persistent difficulty which had eluded treatment prior to the administration of diphenylhydantoin sodium and the calcium-bromine preparation.

CASE 5.—R. Van O., a youth aged 19 years, was referred by Dr. Raymond Katzen on Nov. 24, 1941 with the history of having had periodic bouts of scintillating scotomas, severe headaches and abdominal pain. These attacks were initiated with scotomas composed of flashes of silvery white flickering light, forming an arc in the visual field. Immediately following this, pain developed over the right eye and in the right temporal region. The headaches were then followed by abdominal pain. These attacks usually began in the morning. Preceding the attack the patient's face became ashen white and his hands blanched. He had five such attacks during the year preceding the examination. In addition to these seizures he had frequently been found to walk in his sleep, of which fact he had been entirely unaware except on several occasions when he had fallen downstairs and regained consciousness as a result of the tumble. At 2½ years of age he had a severe head injury with the formation of a hematoma over the right side of the forehead. Scintillating scotomas began at the age of 4. During the past year the attacks had always been accompanied by the abdominal pain, which invariably followed the headaches. These spells lasted from several minutes to as much as one or two hours.

Both parents were living and in good health. His mother, in her youth, was a somnambulist. A maternal grandfather likewise was given to sleepwalking. Two siblings were well and did not suffer from a similar condition. No member in the immediate family or collateral members had headaches or a similar condition.

The patient was cooperative, was well developed physically and showed no evidence of endocrine dysfunction. Examination of the cranial nerves revealed that they were entirely normal. There were no defects of motor dynamics. Tendon reflex activity throughout the body was normal and equal. Pathologic reflexes could not be brought out. There was no disturbance in cerebellar function. Sensation was preserved throughout the body in all modalities. Other phases of the neurologic examination were entirely normal. Blood pressure was systolic 120, diastolic 68; pulse rate 72. X-ray studies of the skull revealed no abnormalities. Complete laboratory studies gave only normal results.

The episodic nature of the attacks, their abrupt onset, their relatively short duration and the associated history of somnambulism prompted me to have an electroencephalogram performed so as to determine whether the entire symptom complex was of a migrainous nature or epileptic in origin. The electroencephalographic studies were carried out by Dr. Joseph Hughes, who reported (fig. 3) that there were slow 5 to 7 per second waves seen throughout both hemispheres, particularly in the frontal and parietal regions and in the left occipital lobe. The rhythms of the right occipital lobe came through at 9 per second. These 5 to 7 per second ranged from 10 to 30 microvolts in amplitude. After a period of hyperventilation, large, slow, abnormal waves varying from 50 to 100 microvolts in amplitude and from 3 to 6 per second in rate appeared in both hemispheres. These were definitely abnormal. This electroencephalographic response was indicative of some type of increased cortical irritability. There was no evidence of any focalized lesion.

The patient had received medication designed to control what formerly had been considered migraine, but to no avail. After the electroencephalographic report indicated presence of abnormal cortical discharges, he was given diphenylhydantoin sodium 1½ grains twice daily, and phenobarbital ¼ grain (0.016 Gm.) three times daily. On May 4, 1942, a little less than six months after the institution of treatment, he developed a mild attack while at work. He had had no somnambulistic excursions, however, during this period. During October 1942 he still had been free from headaches, abdominal pain and scintillating scotomas. He had gained 8 pounds (3.6 Kg.) and was feeling better than he had for many years. During that month he was anxious to enlist in the armed services and, contrary to the advice of his physician and parents, enlisted without informing the induction board of his symptoms and the electroencephalographic findings. He entered the Air Cadet Service and for a time continued to take his medicine without the knowledge of the officer in charge. When the supply of medication was gone he began to develop attacks of exactly the same nature as those of which he originally had complained. He was studied by the medical examiners of the Army Air Force, resulting in his discharge from the Army on a medical certificate.

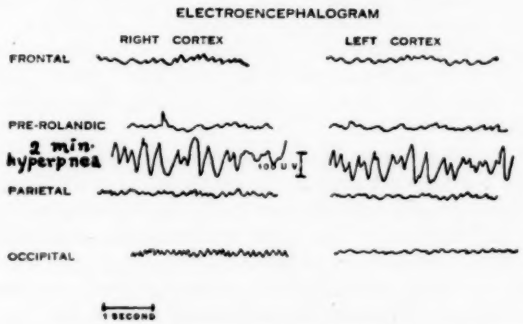

Fig. 3.—Electroencephalographic tracing in case 5.

This patient had formerly been treated for migraine. However, careful evaluation of the symptoms and the associated sleepwalking, together with the history of head trauma during childhood, made it imperative to rule out the possible existence of the convulsive state as the correct explanation. It has been suggested by Wilson [8] that scintillating scotomas and headaches may be epileptic variants and are to be differentiated from true migraine. Gowers,[19] Oppenheim,[20] Riley [21] and Cobb [12] have indicated the probable relationship between migraine and the epileptic state and the need for distinguishing one from the other. Somnambulism has been considered as an epileptic variant. The abdominal pain in this patient occurred as part of the constellation of scintillating scotomas and headache. The electroencephalograms showed definite evidence of abnormal cerebral discharges, the disturbance appearing mainly in the frontal and parietal areas.

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CASE 6.—Mrs. P. P., aged 44, referred by Dr. Lynn Rankin on May 4, 1944, gave as her chief complaint abdominal pain. She stated that her present condition dated back twelve years. Ten years before, because of repeated abdominal pain, she had her appendix removed, the biopsy of which showed no morbid change. The symptoms continued. During the past ten or twelve years she consulted numerous physicians of various specialties without being relieved by many forms of medication and treatment. The individual attacks consisted of what she described as a "gnawing" disturbance followed by generalized pain in the abdomen, accompanied by a "rumbling noise in the intestine." Occasionally the abdominal pain was accompanied by a "funny, dazed feeling," and she often feared that she was going to "pass out." She had never lost consciousness during these spells, however. The attacks occurred mainly at night. On three occasions, all of which occurred while in bed, the attacks of abdominal pain were associated with "shaking" of her limbs and jaws which she was unable to control, the entire episode lasting from ten to fifteen minutes. These attacks sometimes awakened her from sleep. She had not complained of headaches, nausea, vomiting or diarrhea preceding, during or subsequent to these attacks. In some of the episodes of abdominal pain she noticed that the sensation would extend into the chest and up into the head. During the four month period prior to examination, the seizures occurred about twice weekly. She had had free intervals lasting not more than one week. She lost about 10 pounds (4.5 Kg.) during the last eight months. The past medical history revealed no evidence of meningitis, encephalitis, head injury or serious illnesses. She had been married nineteen years, and her husband was living and well. There was no similar condition in the immediate or collateral members of the family.

The neurologic examination was entirely negative. There were no overt disturbances in her emotional or mental spheres. She appeared to be well adjusted in her marital life, and there were no difficulties in her social environment. Examination of the chest and abdomen showed no evidence of gross visceral disease. Previous x-ray studies of the gastrointestinal tract showed no morbid changes, and laboratory studies were entirely normal. Electroencephalographic studies done by Dr. Joseph Hughes showed no abnormal cerebral discharges of the type usually associated with the convulsive state. It was felt, in view of the nature of the seizures, together with the description of the "funny, dazed feeling," the occasional ascension of the abdominal pain into the chest and head similar to the aura preceding a typical grand mal fit and the pseudoconvulsive movements which had occurred on three occasions, that the abdominal pain was a form of abdominal epileptic variant. Accordingly she was placed on anticonvulsant therapy consisting of diphenylhydantoin sodium 1½ grains twice daily and phenobarbital ¼ grain three times daily. Medication was started April 24, 1944, and one week later she stated that there had been a cessation of the abdominal pains and a disappearance of the belching, and that she had improved generally. She has continued to remain on treatment and to date has not had a return of symptoms.

The physician who referred this patient stated that previous examiners had labeled her as a psychoneurotic, after failure to discover an organic cause for her symptom of abdominal pain. The irregularity of the attacks and their nocturnal appearance, aura-like sensations and myoclonic movements on three occasions indicated the epileptic nature of the spells of abdominal pain. The "rumbling noise," described in exactly this way by other patients, may be the auditory representation of increased intestinal activity.

This patient had a normal appendix removed, as did patient 1.

COMMENT

The 6 cases presented here have as a common denominator paroxysmal attacks of abdominal pain. In 2 cases (1 and 6) this happened practically in "pure" form. In case 4 the predominant seizures consisted of abdominal pain occurring in wavelike attacks which corresponded closely to the seizures described in cases 1 and 6. These three cases (1, 4, 6) had been studied quite intensively before the tentative diagnosis of abdominal epilepsy was entertained; 2 of these patients showed abnormal electroencephalographic findings characterized by slow waves occurring mainly in the frontal and parietal lobes. All 3 of these patients responded immediately and continuously to anticonvulsant therapy in the form of diphenylhydantoin sodium and bromides or phenobarbital. Patient 2, during a six month period preceding admission to the hospital, had seizures characterized predominantly by abdominal pain. The latter had been thought to be so-called enterocolitis; treatment in this direction failed to yield results. A careful evaluation of the history in terms of a head injury, periodic bouts of abdominal pain, diarrhea, cold sweats, weakness, occasional loss of consciousness, falling to the floor, convulsive movements of the arms and legs and frothing at the mouth all indicated the probable epileptic nature of the recently outstanding abdominal symptoms. Despite the negative electroencephalographic studies anticonvulsant therapy was begun, and during the time the patient cooperated and took medication he was seizure free.

It will be seen that 3 of the 6 cases had a history of antecedent head injury. One presumably had cerebral angioneurotic edema following the injection of diphtheria antitoxin, with a sensitization or morbid change in the cerebral cortex, and 1 case tuberous sclerosis with undoubted morbid changes in the brain. Case 6 showed no etiologic background. Thus the majority of cases presented give historical evidence of disturbed cortical structure and function.

In view of the experimental and clinical data previously referred to [22] that disturbances of gastrointestinal motility may be influenced by lesions involving the frontal lobe (area 6 of Brodmann) and the parietal lobe (areas 5 and 3), it may be argued that the attacks of abdominal pain experienced in the cases reported here was a manifestation of abnormal discharges occurring in the cerebral cortices, producing intestinal activity of a painful variety. The electroencephalographic findings in 3 of the patients seem to corroborate such a stand. Wechsler [23] observed in his study of abdominal pain as a symptom occurring in space-taking lesions of the brain that "most of the evidence presented points to the cortex and possibly to the frontal more particularly the premotor area as the source of neurogenic abdominal pains."

The 5 patients who were given anticonvulsants responded favorably and quickly to the use of diphenylhydantoin sodium alone or in combination with phenobarbital or bromides. Since the introduction of diphenylhydantoin sodium by Merritt and Putnam [24] into the armamentarium of anti-convulsant drugs it has

22. Fulton,[1] Watts and Fulton.[2] Watts.[3] Sheehan.[3] Spiegel, Weston and Oppenheimer.[3]

23. Wechsler, I. S.: Abdominal Pain as a Symptom of Disease of the Brain, J. A. M. A. **105**: 647 (Aug. 31) 1935.

24. Merritt, H. H., and Putnam, T. J.: Sodium Diphenyl Hydantoinate in the Treatment of Convulsive Disorders, J. A. M. A. **111**: 1068 (Sept. 17) 1938.

proved one of the most valuable in this field. Goldman [18] has studied the effect of diphenylhydantoin sodium and phenobarbital on the electroencephalograms of patients suffering from severe epilepsy and has shown the damping out effect, by these anticonvulsants, of the abnormal cerebral discharges. This effect on the electroencephalogram was demonstrated in cases 1 and 4, the only 2 in which electroencephalographic studies were performed before and during treatment.

One half of the 6 cases reported here gave a history of head injury. This, as a possible cause of abdominal epilepsy,[17] "is considered important at this time because of the strong likelihood of many cases of epilepsy or its variants finding their way into medical practice as the result of craniocerebral injuries and infections incident to World War II, and those which may appear with the return of the automobile and airplane to extended civilian use."

To establish the diagnosis of abdominal epilepsy and its etiologic background, it is necessary, in those patients presenting the symptom of abdominal pain, to observe the following steps in study:

1. Cases must be examined intensively, by all available methods, to rule out intrinsic disease of the pelvic, abdominal and thoracic viscera, morbid involvement of the spinal cord or peripheral nerves and psychosomatic projection mechanisms.

2. Historical data regarding the possible existence of cerebral disease or dysfunction should be uncovered.[12]

3. The behavioristic occurrence of the pain should adhere, in major respects, to the pattern of epilepsy so aptly stated by Wilson:[25] "Any fugitive, paroxysmal, disorderly, uncontrolled and recurrent manifestation of any neural process might be regarded as a fit, and this on any level of the neuraxis. . . . As for fits implicating visceral centers, there may be no interference with the conscious stream, no involvement of skeletal muscles and, in fact, none of the usual signs of epilepsy, so that divergence of the formula may be extreme. . . . The least mutable of accepted fit characters comprise sudden onset, brief duration and recurring habit; yet some forms are protracted (notably epilepsia partialis continua), repetitions may be extraordinarily infrequent, and brevity loses significance when the aura (as in some uncinate cases) is drawn out."

4. The presence of associated epileptic phenomena, either preceding or during the presenting episodes of abdominal pain, will form corroborative evidence of the nature of the disorder.

5. Positive neurologic signs and laboratory findings including cytologic and chemical studies, roentgenologic studies of the skull and electroencephalographic studies, indicating the existence of a cerebral structural or physiologic disorder, should be in evidence.

6. Salutary effects of anticonvulsant drugs on the occurrence of abdominal pain should be exhibited, and the influence of these drugs on the electroencephalogram should in most instances parallel the clinical results.

SUMMARY

Clinical study of 6 patients (2 children, 4 adults) in whom paroxysmal abdominal pain was either the only symptom or the dominant one revealed this to be a type of epileptic variant (abdominal epilepsy), as shown by the pattern of symptomatology, electroencephalography and response to anticonvulsant drugs. Three of the patients presented the history of antecedent head injury; 1 had cerebral angioneurotic edema following injection of antiserum prior to the onset of abdominal seizures; 1 had paroxysmal abdominal pain diagnosed as intestinal tetany occurring in an unrecognized case of tuberous sclerosis; 1 showed no etiologic background.

There is need for recognition of unexplained paroxysmal abdominal pain, when study of the abdominal viscera has been negative, as being possibly of cerebral origin due to abnormal cerebral discharges arising in the premotor or parietal areas. These symptoms are liable to be present in the returning war veteran who has suffered a severe head injury, and there is likelihood of failure to evaluate its true nature.

The criteria for diagnosis are based on (1) exclusion of intrinsic visceral disease, (2) adequate historical data, (3) attack pattern of an epileptic order, (4) associated epileptic show, (5) objective evidence of cerebral organic disease or dysfunction and (6) effect of anticonvulsant drugs on the symptom of abdominal pain and on the electroencephalogram.

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25. Wilson: Neurology,[16] pp. 1469-1470.

ABSTRACT OF DISCUSSION

DR. STANLEY COBB, Boston: Dr. Moore is to be congratulated on his excellent clinical paper. The 6 cases are clearly presented and are convincing. I agree in his diagnosis and consider that his careful study should be taken as a model. I especially like his emphasis on history and the clinical descriptions of the seizures. The success of therapy with anticonvulsant drugs is another impressive part of the evidence which builds up his diagnosis. In case 2, in which the history is clearly that of epilepsy, there is a normal electroencephalogram. The author quotes Gibbs as saying that such normal tracings may occur in 8 per cent of patients with post-traumatic epilepsy. My experience is that many more than 8 per cent have normal electroencephalograms between attacks. If caught in an attack, of course practically every patient shows abnormal discharges. A normal tracing between attacks does not rule out epilepsy. On the other hand, the finding of abnormal waves is important evidence in favor of this diagnosis when the history and clinical description lead one to suspect epilepsy. I do not like the use of the terms "petit mal," "grand mal" and "psychomotor," as if all kinds of epileptic seizures could be classified under one of the headings. In a consulting practice one sees more "atypical fits" or "variants" than orthodox convulsions. Variations in consciousness with peculiar behavior are common and more important to remember than the textbook picture and "grand mal," in which the sequence of events is said to be aura, unconsciousness, tonic spasm and clonic jerks followed by stupor. Common enough, I grant, but any street car conductor can make the diagnosis in such cases. Dr. Moore merits our thanks for describing a variant that is not rare and is often wrongly diagnosed. Case 4 illustrates the point that "grand mal" and "petit mal" are not as useful terms as one is given to suppose. The fits are spoken of as "abortive grand mal," and certainly more of them were very far from being typical. The electroencephalogram showed "groups of spike and slow wave forms," which Lambert (quoted earlier in the paper) would call a dysrhythmia of the "petit mal type." The history of this patient clearly shows that he never had any fits remotely resembling "petit mal." In short, types of electroencephalographic wave and types of clinical fit do not correspond very well, and there is still much to be learned in this field. It may be of interest to point out that in 3 of these cases, to a striking degree, and in all of them to some extent, the symptoms resemble those of neostigmine poisoning. What could this mean in relation to epilepsy, the autonomic nervous system and acetylcholine?

DR. TRACY PUTNAM, New York: Dr. Moore has clearly brought out the characteristics and significance of the abdominal pain and other digestive disturbances which may occur as a form of epileptoid attack. In my experience such attacks are particularly common and puzzling in children, and I have studied a large number of cases since the original publication of Klingman, Langford, Greeley and Hoefer from this clinic. The occurrence of such attacks following head injury is of great interest and importance. I am particularly happy to hear his criteria for diagnosis so clearly enunciated. The boundaries of the still poorly explored territory of the epileptoid states are not clearly marked, and such definitions as he has formulated will do much to clarify them. If they are rigorously applied to other puzzling syndromes without obvious gross structural basis, and of paroxysmal nature, it is likely that we shall find still other new epileptic equivalents.

DR. E. A. SPIEGEL, Philadelphia: Dr. Moore's observations are a beautiful example of clinical application of experimental results. Fulton and his colleagues observed changes of gastrointestinal activity on stimulation of the so-called premotor area of the frontal lobe, while our group demonstrated that such effects could also be obtained from "postmotor" foci in the parietal lobe. We were also able to trace, by means of Marchi degeneration, descending fibers from these postmotor areas, which fibers joined the pyramidal tract system. Dr. Moore's observations corroborate these findings in that some of his cases showed abnormal discharges not only from the frontal but also from the parietal lobes. He is inclined to interpret the attacks of abdominal pain in these patients as due to gastrointestinal spasms elicited by impulses descending from these areas of abnormal cortical discharges. Such an interpretation seems justified in the majority of his material, in that signs of abnormal gastrointestinal activity were demonstrable during the paroxysmal abdominal pain. However, besides the stimulation of descending systems acting on the gastrointestinal tract, pain projected into the abdomen could also be produced by a somewhat different mechanism, namely by stimulation of cortical sensory areas receiving impulses from the viscera. Dr. Hunsicker and I observed changes of the electrocorticogram on stimulation of afferent fibers of the pelvic nerve, and Bailey and Boemer made similar observations for afferent vagal fibers. It seems to me that stimulation of such cortical sensory areas for afferent impulses from the viscera could play a role in cases like the sixth of this series, in which the sensation extended into the chest and up into the head.

DR. N. W. WINKLEMAN, Philadelphia: Evidence is gradually accumulating that the cerebral cortex is concerned with the motility of the gastrointestinal tract. Gross lesions, particularly of the frontal lobes, have been shown to produce abnormal intestinal movements. It may well be that hypermotility of the gastrointestinal tract on a known emotional basis may result from stimuli that also originate in the same cortical region. As an illustration of the first group, a man aged 45, whose initial symptoms were abdominal pains of such severity that he was actually "doubled up" gradually became stuporous and died within a period of four to five days. At autopsy a softened area in the fronto-motor-parietal area was found, into which hemorrhage had occurred. The 6 cases reported by Dr. Moore are interesting and instructive. In case 1 the temporal relationship of the first attack to the diphtheria antitoxin is somewhat vague, yet the patient must have been allergic in view of urticaria at a later date. This brings up the question of the effect of the antitoxin on the brain. Go'ten and I (Encephalomyelitis Following the Use of Serum and Vaccine, *Am. J. Syph. & Neurol.*, **19**:414 [July] 1935) have shown that an acute encephalomyelitis disseminata can develop in susceptible individuals from serums and vaccines. Permanent cerebral damage can result. I am sure that most of us would have first diagnosed migraine in case 5. The history of attacks of hemicrania preceded by scintillating scotomas is certainly suggestive of this condition. The history of somnambulism, however, suggests an added condition—epilepsy. Somnambulism can be a postepileptic automatism as well as an epileptic equivalent. Migraine and epilepsy are not unknown in the same patient and may be related, as some believe. The abdominal pain in this case, therefore could have been either abdominal migraine or abdominal epilepsy. Dr. Moore told me that he too treated this patient for migraine before realizing that it was part of the epileptic syndrome. It was the natural thing to do. In the other cases the relationship between convulsions and the abdominal crises is more direct and more definite. Treatment for the convulsions in these cases should relieve or ameliorate the abdominal symptoms. The lesson taught by this paper is obvious. The nervous system can and does exert influence at a distance, and many apparently remotely removed symptom complexes may have as their basis some organic problem within the central nervous system.

DR. MATTHEW T. MOORE, Philadelphia: I wish to express my thanks and indebtedness to the discussers for their valuable and pertinent comments. I am completely at one with Drs. Cobb and Putnam regarding the epileptoid states and the nosologic inadequacy of such terms as "grand mal," "petit mal" and "psychomotor." These terms represent the mental conditioning of the physician as a means of orientation, but it must be stressed, as Dr. Cobb properly does and as I have done elsewhere,[17] that "atypical fits" or "variants" occur more frequently than orthodox epileptic convulsions. Moreover, they generally go unrecognized because, in Dr. Putnam's words, "the boundaries of the still poorly explored territory of the epileptoid states are not clearly marked," which poses the challenge to us of delineating more clearly these boundaries and of corralling the many vagrant forms into a recognizable group. Dr. Cobb's observation regarding the similarity of the abdominal symptoms to neostigmine poisoning is extremely interesting, since this thought had occurred to me at the time I was studying patient 1. The implications of this observation should be further explored experimentally in terms of the role played by the autonomic nervous system and the ultimate physiobiochemical mediators or effector agents which may produce the manifold expressions of the epileptic state. It is true, as Dr. Spiegel states, that abdominal pain, or pain projected to other visceral areas, may be due to stimulation of the cortical sensory areas receiving afferent impulses from the viscera, but this can occur with an unaltered normal cortex. This mechanism of afferent impulses stimulating either motor or sensory areas or both probably is the explanation for the phenomenon of so-called reflex epilepsy. However, in the majority of the cases reported here it is presupposed, on the basis of the history, neurologic data and electroencephalographic studies, that the abnormal discharges arise in the vicinity of disturbed cortical tissue, albeit these areas may be fired off by remote afferent impulses of the order described by Dr. Spiegel or by humoral factors. The case cited by Dr. Winkelman is a striking example of a gross cerebral lesion causing abdominal pain. It will be noted that both the frontal and parietal lobes were involved. With such material and the cases reported here as a basis on which to work, is it not likely, therefore, that many of the unexplained and commonly occurring attacks of abdominal pain may be due to abnormal discharges arising from sensitized, traumatized or morphologically altered areas of the frontal and/or parietal cortex?

Alcohol and Accidents.—Alcoholic intoxication is one of many factors contributing to traffic accidents. The factors contributing to accidents may be classified in the following four general categories: (1) driver factors, (2) road factors, (3) vehicle factors and (4) pedestrian factors. Driver factors are especially important and are made up of three classes: deficiencies in physical condition, lack of knowledge or skill and improper attitude. Physical deficiencies include not only permanent physical deficiencies, such as a lost arm or leg, but also such temporary deficiencies as those resulting from fatigue or the influence of alcohol. Many drivers with permanent physical deficiencies compensate for them, but it is difficult to compensate for a temporary deficiency such as the influence of alcohol.—Berry, Donald S., in Alcohol, Science and Society, New Haven, Quarterly Journal of Studies on Alcohol, 1945.

CLINICAL TRIALS WITH QUININE-EPINEPHRINE INTRAVENOUSLY

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The successful current use of quinacrine (atabrine), orally and intramuscularly, in treating malaria has not relegated quinine to the scrap-heap of obsolete medicinals for this infection. Quinine has been widely used intravenously in civilian practice and in the armed services as an emergency measure in, or for initiating treatment of, the embolic or cerebral form of the disease, especially with coma. The effectiveness of the quinine, under these conditions, has not been challenged, but the safeness of the procedure has been considerably debated.

In general, clinical reports [1] have been rather opinionated without substantial support of the claims for or against using quinine intravenously. The possible hazard in the circulatory depression from quinine has been consistently recognized, although the intravenous procedures used have not always considered measures for controlling or combating this. The alkaloid has been given empirically with or without an antagonist. Intravenous epinephrine has been condemned by some almost as much as quinine. However, use of an appropriate antagonist with the quinine would seem to be desirable, at least, in depressed circulatory states, as in comatose patients with the embolic type of malaria.

Since nothing has been known of the scope of possibly desirable antagonists, Dreisbach and Hanzlik [2] made an extensive experimental study of this problem in several directions. Of some thirty-two agents tested, three were found to be effective against doses of quinine of therapeutic order. These were epinephrine, neosynephrine and calcium chloride, in descending order of efficiency and desirability. Epinephrine and neosynephrine were also the most effective antidotes of a number of those commonly recommended in severe circulatory collapse after high doses of quinine. In animals it made practically no difference, according to the percentage fall in blood pressure, whether the quinine was injected comparatively rapidly, or slowly, as for thirty minutes, advised clinically. Experimentally the choice agent was epinephrine, 0.015 mg. per kilogram of body weight, mixed with quinine sulfate 10 mg. per kilogram in isotonic solution of sodium chloride (U. S. P.). This dose of epinephrine prevented or mitigated the depressor action of the quinine, whereas 0.01 mg. per kilogram was not always adequate, and 0.02 mg. per kilogram considerably raised the blood pressure. The antagonistic action of the epinephrine generally was not successful after the third dose of quinine.

Translated to a 70 Kg. man, and assuming similar actions, the dose of 0.015 mg. of epinephrine per kilogram was the equivalent of a total dose of 1.05 mg., which agreed almost exactly with 1.0 mg. recommended by Escher and Villequez [3] in 1931. The published report of these French army medical officers has been generally overlooked by others. As the result of an extensive experience with different methods of administering quinine in malaria in the tropics, Escher and Villequez recommended the following mixture for intravenous injection, especially for patients in shock or coma: 0.5 Gm. (up to 0.8 Gm.) of quinine hydrochloride and 1 mg. of epinephrine in 250 cc. of isotonic solution of sodium chloride injected in not less than thirty minutes. Escher and Villequez favored the general use of quinine intravenously in malaria as being the most effective medication and used it without epinephrine for noncomatose patients.

Practically in agreement with Escher and Villequez is a recent clinical report by Fitz-Hugh, Pepper and Hopkins [4] which appeared after the experimental results of Dreisbach and Hanzlik were submitted for publication. These United States Army medical officers treated 6,059 patients with malaria in India, and of these 140 had cerebral malaria. They found that the great majority of patients with cerebral malaria required one or more intravenous injections of quinine dihydrochloride, 0.32 to 0.65 Gm., in saline or saline-glucose solutions, before they could be aroused enough to take oral medication. The drug was slowly administered and well diluted (200 cc. minimum—preferably 500 cc. or more) and caused no fatalities fairly attributable to its use. As to the value of epinephrine and of quinine intravenously and the precautions to be observed, they state that the intravenous administration of epinephrine (1 cc. of 1:1,000 solution well diluted) proved of value for certain comatose patients; if well tolerated it may be repeated, on relapse into coma, by venoclysis in isotonic solution of sodium chloride or saline solution with or without quinine dihydrochloride. Epinephrine, however, is not to be used routinely and is probably contraindicated in shock and pulmonary edema. The blood pressure response should be watched carefully and the administration discontinued if the blood pressure rises above 160 or the pulse rate is much accelerated or if other untoward symptoms appear. Intramuscular or oral atabrine may be used to supplement quinine, but they say that they have more confidence in quinine dihydrochloride administered intravenously, supplemented as soon as possible by oral quinine, than in any other specific in these cases.

More recently, MacDonald [5] of the British army has advised combined operations in attacking malaria by using epinephrine intravenously to drive parasites out into the open and quinine at the same time orally, subcutaneously or intravenously. Marsh [6] has stated that quinine and epinephrine given intravenously are usually necessary in the treatment of cerebral malaria, although prophylaxis with quinacrine daily will almost always prevent blackwater and cerebral malaria. Although the statements by Fitz-Hugh, Pepper and Hopkins summarize the essential features of using quinine intravenously in malaria, neither these authors nor

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1. Brahmachari, U. N.: Dangers of Rapid Intravenous Injection of Quinine Solution, Lancet **2**: 175 (July 22) 1922; Dangers of Rapid Intravenous Injection of Concentrated Solutions of Quinine Bihydrochloride, J. Trop. Med. **25**: 209 (July) 1922; A Note on Blood Pressure During Intravenous Injections of Quinine, Lancet **2**: 1301, 1920. Simpson, W. M., and Sagebiel, J. L.: Cerebral Malaria: A Report of 12 Cases Encountered at a U. S. Naval Base Hospital, U. S. Nav. M. Bull. **41**: 1596 (Nov.) 1943. Maxcy, K. F.: Limitations to the Use of Quinine Intravenously in Treatment of Malaria, Pub. Health Rep. **37**: 693 (March 24) 1922; The Status of Intravenous Therapy: V. Limitations to the Use of Quinine Intravenously in the Treatment of Malaria, J. A. M. A. **91**: 1372 (Nov. 3) 1928. Brosius, O. T.: Estivoautumnal Malaria, ibid. **125**: 168 (May 13) 1944. Most, H., and Meleney, H. E.: Falciparum Malaria: The Importance of Early Diagnosis and Adequate Treatment, ibid. **124**: 71 (Jan. 8) 1944.

2. Dreisbach, R. H., and Hanzlik, P. J.: Antagonists for the Circulatory Depression of Quinine Injected Intravenously and the Implied Cholinergic Action, and Nature and Importance of the Vasodilatation, in the Depression, J. Pharmacol. & Exper. Therap. **83**: 167, 1945.

3. Escher and Villequez: Intravenous Injection of Quinine in Malaria, Presse méd. **39**: 453 (March 28) 1931.

4. Fitz-Hugh, T.; Pepper, D. S., and Hopkins, H. U.: The Cerebral Form of Malaria, Bull. U. S. Army M. Dept., 1944, No. 83, p. 39.

5. MacDonald, D. C.: Adrenalin in the Treatment of Malaria, Brit. M. J. **1**: 567 (April 21) 1945.

6. Marsh, Frank: Treatment of Cerebral Malaria, Brit. M. J. **1**: 641 (May 5) 1945.

Escher and Villequez report details of possible circulatory and other changes in their patients, and MacDonald and Marsh give neither such details nor the dosage of drugs. These are common deficiencies in other clinical reports.

Therefore it was believed desirable to obtain some information on the possible general effects and tolerance of clinical intravenous injection of quinine-epinephrine, partly for confirmation or refutation of the clinical claims and partly to obtain familiarity with it in the event that such injection might be indicated in treating malaria. Safeness of the procedure was an important consideration. Such trials were made by one of us (C. C. C.) and his associates with 7 male patients from 18 to 48 years of age. In 3 of these (M. P., H. H. and C. W.) the initial circulatory state was about normal, although M. P. had a high pulse rate. In a fourth patient (C. A.) the initial blood pressure was above normal, the heart rate being about normal. Three other patients were in coma with circulatory depression, as the result of acute intoxication from alcohol, pentobarbital and paraldehyde. One injection of the following mixture was made into the median basilic vein in each patient: 0.5 Gm. of quinine bisulfate and 1.0 mg. of epinephrine hydrochloride in 250 cc. (5 patients) or 300 cc. (2 patients) during a period of thirty minutes. Blood pressure, pulse rate and respiration were observed before and at the end of fifteen minutes and of thirty minutes after the injection was begun. All patients were observed for at least twenty-four hours afterward. A summary of the essential data is presented in the accompanying table.

Clinical Effects of Quinine-Epinephrine Intravenously

			Quinine Bisulfate 0.5 Gm. plus Epinephrine 1.0 Mg. Intravenously						
			Before Injection		15 Minutes After Injection		30 Minutes After Injection		
Patient	Age, Yrs.	Diagnosis	Blood Pressure§	Heart Rate, per Min.	Blood Pressure	Heart Rate, per Min.	Blood Pressure	Heart Rate, per Min.	Other Effects
M. P.*	48	Hepatic cirrhosis	120/ 60	120	120/ 60	125	120/ 60	120	None
H. H.*	45	Toxic psychosis	120/ 70	80	120/ 70	80	120/ 70	80	None
C. W.	28	Spinal cord transection; pneumonia; apprehension; temp., 104 F. (Negro)	110/ 60	76	150/ 80(1)†	88	175/ 76(20)†	110	Restlessness, suffocation, dyspnea, screaming in 1 min., quiet but flushed in 20 min., recovered in 45 min. after injection
					150/ 70(15)†	96	120/ 80(45)†	86	
C. A.	46	Nephrosis	190/105	88	200/100	96	190/105	88	None
P. F.	24	Acute paraldehyde intoxication deep coma	80/ 50	90 (Resp. 8)	90/ 60	110 (Resp. 8)	100/ 60	90 (Resp. 8)	Coma unchanged; after 2 doses of metrazol within ½ hour after quinine-epinephrine consciousness regained; blood pressure 120/60, with complete recovery in a few hours
P. F.	27	Acute alcoholism; possible subdural hematoma; coma	60/ 45	90 (Resp. 10)	82/ 50	115 (Resp. 18)	80/ 50	88 (Resp. 20; Temp. 98.6 F.)	Coma unchanged; complete recovery following morning
R. S.	18	Sodium pentobarbital intoxication; deep coma	100/ 70	90 (Resp. 10)	100/ 80	94 (Resp. 10)	100/ 75	86 (Resp. 10)	Coma unchanged; complete recovery in 24 hours

* Quinine-epinephrine in 300 cc. of isotonic solution of sodium chloride; in 250 cc. in the remaining patients.
§ Systolic/diastolic in mm. Hg throughout. † Minutes after injection.

It is seen that the most consistent change was an increase in pulse rate fifteen minutes after the injection was started, i. e. from about 4 to 27 per cent in 6 of 7 patients. At the end of the injection (thirty minutes) the pulse rate had returned to the initial rate in all but the most excitable patient (C. W.). The systolic blood pressure was appreciably increased in 4 of the patients, i. e. about 5 per cent in C. A., in whom the initial pressure was the highest, 12.5 per cent in P. F. in paraldehyde coma and 37 per cent in P. F., in alcoholic coma, and 50 per cent in C. W., initially excited. Again these increases occurred at the end of fifteen minutes, with recoveries practically to the initial levels at the end of thirty minutes. The diastolic pressure was also increased at the end of fifteen minutes in 4 of the patients, i. e. from about 16 to 33 per cent in excitable C. W., about 20 per cent in P. F. (paraldehyde), about 11 per cent in P. F. (alcohol) and about 14 per cent in R. S. (pentobarbital). Recoveries were incomplete at the end of thirty minutes. Three patients (M. P., H. H. and R. S.) showed no demonstrable changes in systolic blood pressure, and 2 (M. P. and H. H.) in diastolic pressure. No other effects were observed in 3 patients (M. P., H. H. and C. A.) and the coma remained unchanged in the 3 (P. F., P. F. and R. S.) who had taken alcohol, paraldehyde and pentobarbital. The most pronounced reaction occurred in the most excitable patient (C. W.), who was apprehensive and excited before the injection began. It is doubted whether the restlessness, suffocation, dyspnea and screaming which occurred one minute later were due to the procedure; complete recovery was present at the end of forty-five minutes. Respiration was variably affected in the comatose patients, being unchanged in P. F. (paraldehyde) and in R. S. (pentobarbital) and increased in P. F. (alcohol). Presumably the rather rapid recovery of consciousness and blood pressure was due to the metrazol in P. F. comatose from paraldehyde. The patients in alcoholic and pentobarbital comas who did not receive this treatment required much longer periods for recovery. Thus, in general, the principal reaction to a single injection of quinine-epinephrine, in the doses used, in conscious and comatose patients was a fleeting circulatory stimulation, principally an increase in heart rate; the blood pressure was moderately, though variably and temporarily, increased in roughly one half of the patients. Since this was confirmatory of the extensive experiences of Escher and Villequez and of Fitz-Hugh, Pepper and Hopkins, further trials were not believed necessary. We had no opportunity of trying the quinine-epinephrine mixture in malarial coma, but the mixture used by us was not detrimental, at least, in comas of drug intoxications.

It is of interest to compare the dosage of quinine, as base, used by us and the other reporters, since the salts used were different with considerable variation in

content of quinine. Generally the safeness of the procedure would vary inversely and its antimalarial efficiency directly as the dosage. On an equimolecular basis the amounts of quinine base in the single doses of the different quinine salts were as follows: 0.477 to 0.762 Gm. in 0.5 to 0.8 Gm. of quinine hydrochloride used by Escher and Villequez; 0.286 to 0.619 Gm. in 0.3 to 0.65 Gm. of quinine dihydrochloride used by Fitz-Hugh, Pepper and Hopkins; 0.345 Gm. in 0.5 Gm. of quinine bisulfate used by us; 0.338 Gm. in 0.7 Gm. of quinine sulfate for a 70 Kg. adult based on the standard dose used in animals by Dreisbach and Hanzlik. Accordingly, our doses, i. e. the clinical tried by us and those based on experimental work by Dreisbach and Hanzlik, which were close, were smaller than those used by the other reporters. Smaller doses would be less hazardous intravenously, but their antimalarial efficiency would also be lower, except that this disadvantage would be offset by a lower toxicity, which would permit injecting more doses. However, there seems to be no doubt that the higher range of dosage used by Escher and Villequez and by Fitz-Hugh, Pepper and Hopkins was used successfully, except that their maximum doses appear undesirable for general recommendation.

RECOMMENDED PROCEDURE

Although we did not test the dose of 0.5 Gm. quinine hydrochloride or dihydrochloride (0.477 Gm. base), partly because of the unavailability of these salts and partly because we were guided by the experimental results of Dreisbach and Hanzlik with a smaller dose, this had been included in the extensive trials of Escher and Villequez and of Fitz-Hugh, Pepper and Hopkins and used previously by others. It is believed to be sufficiently established and safe to be worthy of recommendation as emergency, or to initiate, medication for comatose patients with the embolic or cerebral malaria. The preferred procedure is as follows: 0.5 Gm. of quinine hydrochloride or dihydrochloride (0.69 Gm. of quinine bisulfate) and 1 mg. of epinephrine in 250 cc. of isotonic solution of sodium chloride injected during not less than thirty minutes. Not more than two or three such injections, or a total of 1 to 1.5 Gm. of quinine, should be given, properly spaced, within twenty-four hours. When consciousness is restored, medication is to be continued by mouth or through a nasal catheter as suggested by Fitz-Hugh, Pepper and Hopkins. The intravenous injection of quinine is probably contraindicated in severe shock, especially hemorrhagic, pulmonary edema, cardiovascular disease, cyanosis, severe anemias, pregnancy and possibly other conditions.

CONCLUSIONS

1. Intravenous injection of quinine has been widely used in the treatment of the embolic or cerebral form malaria with coma, but the safeness of the procedure has been considerably debated.

2. Experimentally it has been established that the circulatory depression accompanying such injections can be effectively antagonized by injecting epinephrine together with the quinine, but data indicating the clinical reactions to such a mixture have been lacking.

3. Trials made by us with a quinine-epinephrine mixture on 7 patients, 3 of whom were in coma from various drug intoxications, resulted in a fleeting circulatory stimulation, chiefly an increase in heart rate, the blood pressure being moderately, though variably and temporarily, increased in roughly one half of the patients. In the majority there were no other demonstrable effects, and the comas were unaffected. These results sustain the claims of proponents of such medication.

4. Accordingly, quinine-epinephrine intravenously is believed to be sufficiently established and safe as emergency, or initial, medication for comatose patients with embolic or cerebral malaria, in agreement with recent practices of military medical services of different countries, which claim further that such medication is essential or indispensable in this type of malaria.

5. The preferred procedure is 0.5 Gm. of quinine hydrochloride or dihydrochloride (or 0.69 Gm. quinine bisulfate) and 1 mg. of epinephrine in 250 cc. of isotonic solution of sodium chloride injected in not less than thirty minutes. Not more than two or three such injections (total 1 to 1.5 Gm. of quinine hydrochloride) should be given, properly spaced, within twenty-four hours. When consciousness is restored, medication is to be continued by mouth or through a nasal catheter.

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CLINICAL TETANUS

A STUDY OF FIFTY-SIX CASES, WITH SPECIAL REFERENCE TO METHODS OF PREVENTION AND A PLAN FOR EVALUATING TREATMENT

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Tetanus continues to be a serious disease in spite of the availability of practical methods for its prevention and the use of improved serums and drugs in its treatment. In 1942 there were recorded 698 deaths in the United States from this disease,[1] and probably the number of cases was two to three times this figure.

Statistical studies dealing with large numbers of cases [2] have not been particularly useful in evaluating the results produced by any given program of treatment. The need for better methods to evaluate the results from the treatment of tetanus has become more urgent than ever because of the advent of purified antitoxins, recently developed anticonvulsant drugs and new chemotherapeutic agents.

Since the outcome of tetanus, either treated or untreated, depends in large measure on the severity of the disease, one must consider this factor in the evaluation of treatment. From an analysis of the clinical features of 56 cases of tetanus treated in the Infants' and Children's Hospitals from 1924 to 1944 it was possible to place the cases in a series arranged according to the severity of the infection. Such an arrangement enables one to ascertain readily the effects of treatment on the expected course of the disease. One of my purposes in this article is to describe the method of analysis that was used and to discuss some of the clinical implications derived from this analysis. The second purpose is to show the desirability of producing and continuously maintaining active immunity against tetanus as disclosed by a study of the initial lesions in these patients.

From the Department of Pediatrics, Harvard Medical School, and the Infants' and Children's Hospital.

1. Vital Statistics of the United States 1942, United States Department of Commerce, Bureau of the Census, 1944, part 1, table 2.

2. Golla, F.: Analysis of Recent Tetanus Statistics, Lancet **2**: 966 (Dec. 29) 1917. Calvin, J. K., and Goldberg, A. H.: Prognosis of Tetanus, J. A. M. A. **94**: 1977 (June 21) 1930. Huntington, R. W., Jr.; Thompson, W. R., and Gordon, N. H.: Treatment of Tetanus with Antitoxin: Analysis of Outcome in 642 Cases, Ann. Surg. **105**: 93 (Jan.) 1937.

METHODS

Each record was studied in its entirety in order to verify the diagnosis and to determine any unusual features. Then an abstract of each case was made covering only the following four features, which have been stressed by numerous investigators as the most useful indexes of the severity of the infection: [3]

1. The incubation period.
2. The length of time from the onset of symptoms to definite episodes of generalized muscle spasms.
3. The severity of the infection as judged by examination of the patient at the time of admission to the hospital.
4. The frequency and severity of spasms or convulsions after sedation, and the amount of sedation required each day.

No notation as to the method of treatment or the final outcome was made in the abstracts in order to eliminate any conscious or unconscious bias in analyzing the cases. Since all the patients were between 5 days and 12 years of age, the age factor was not included for analysis.

The abstracts were studied, using the four criteria mentioned previously, and the most weight was given to the length of time from the first symptoms until definite episodes of generalized muscle spasms occurred, and to the frequency and severity of spasms after sedation. Then the cases were arranged in a series from the most severe to the mildest. Several months later, using the same abstracts, the cases were again graded as to severity, and no case differed in rank by more than three from its previously assigned position. Thus the reliability of the method was demonstrated.

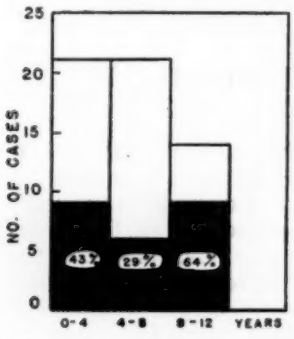

Fig. 1.—Distribution of cases and deaths by age groups.

RESULTS

Fifty-six cases were reviewed. There were 24 deaths, yielding a gross mortality rate of 43 per cent. The distribution of the cases and of the deaths in three age groups is shown in figure 1. There were 39 males and 17 females, with mortality rates of 46 per cent and 35 per cent respectively. Nineteen patients were classified as having severe, 12 as having moderate and 37 as having mild generalized tetanus. Three patients had only local tetanus. In only 1 patient was the diagnosis doubtful. This patient, who died, is included in all figures, since treatment for tetanus was given.

The incubation period in patients classified as having severe generalized tetanus varied from five to ten days, with an average of seven and a half days. (The incubation period was known in 12 of these 19 patients.) The duration from the onset of symptoms to definite episodes of generalized spasms in these patients averaged one day, with a range from one-half to two and a half days. All these patients had pronounced trismus, generalized increase in muscle tone and great hyperirritability, with easily induced spasms or convulsions on admission, and all had severe spasms and 75 per cent had convulsions in spite of large doses of sedatives.

For patients with moderately severe tetanus the incubation periods were not definitely known, except for nine days in 2 patients. Generalized spasms occurred from one to three and a half days, with an average of two and a half days, after the onset of symptoms. All had trismus, increased muscle tone and hyperirritability on admission, and after moderate to large doses of sedatives 6 patients had convulsions and all except 1 had severe spasms. In the patients with mild infections the progress of symptoms was slower and the severity of the spasms was less than in patients with severe and moderately severe tetanus.

The end result, the total amount of antitoxin and its routes of administration, the chief and contributing causes of death and the duration of hospital stay for 37 patients are shown in figure 2. In this chart the cases are arranged in order of decreasing severity of the disease. The 19 patients not shown had mild nonfatal infections. Only 1 of these 19 patients received antitoxin intrathecally, and that patient had no reaction from the therapy. Although the order in which the cases in figure 2 were arranged was made without reference to the final outcome, it is interesting to note that, irrespective of the treatment given, for the first 25 patients death was the general outcome, whereas nearly every one of the patients with order numbers higher than 25, that is, with milder infections, recovered.

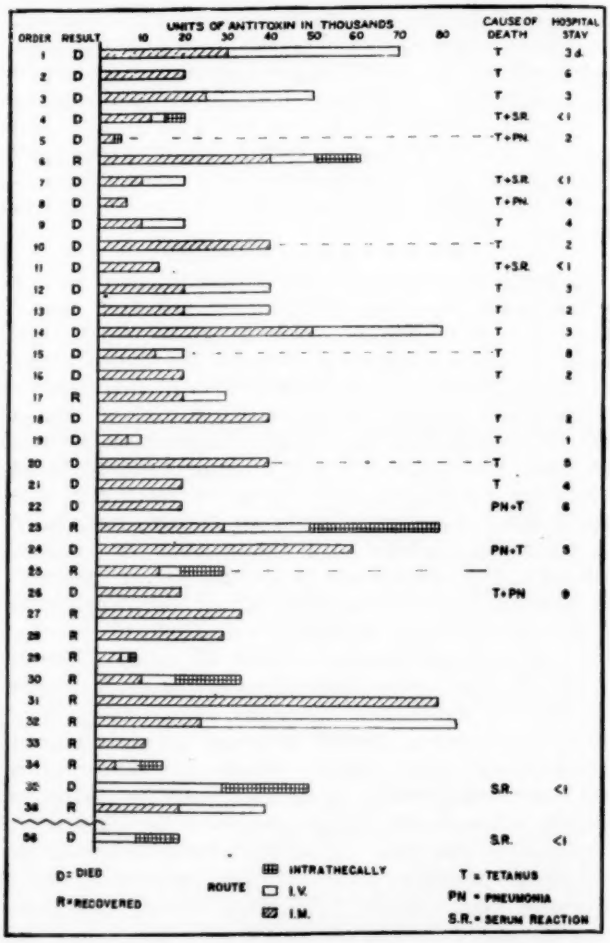

Fig. 2.—Cases of tetanus arranged in order of decreasing severity.

COMMENT

The Value of Arranging Cases in Order of the Severity of the Infection.—A reliable index of the relative severity of the disease can be obtained only by comparing several clinical features of the illness in any given patient with the same features in previous patients. In order to establish the rank of one patient with respect to

3. Huntington, R. W., Jr.: Treatment of Tetanus, Yale J. Biol. & Med. **3**: 207 (Jan.) 1931. Spaeth, R.: Clinical Study of Tetanus, Am. J. Dis. Child. **60**: 103 (July) 1940. Firor, W. M.: Tetanus, in Cecil, R. L.: A Textbook of Medicine, ed. 6, Philadelphia, W. B. Saunders Company, 1943, p. 193.

others in the series, knowledge of the individual records is essential. Therefore, exact comparisons between series prepared in different clinics would not be possible. However, such an arrangement is practically the only useful aid in evaluating the various methods of treatment employed in any given clinic and in planning individualized therapy for each patient.

It is clear from a study of figure 2 that improvement in treatment can be confirmed by the results obtained only in patients with severe infections, that is, in patients classified with those whose infections previously were generally fatal. The superiority of a new method of treatment would be demonstrated if it enabled several patients to survive infections as severe as those in the first 25 patients shown in figure 2.

The Amount of Tetanus Antitoxin.—Study of figure 2 revealed no evidence that amounts of antitoxin up to 80,000 units were more effective than 30,000 units. In accord with this observation are the immunologic data of Spaeth.[4] However, Vener and Bower,[5] who reported 100 cases of tetanus with the low gross mortality rate of 29 per cent, advised a minimum of 200,000 units of tetanus antitoxin.

Route of Administration of Antitoxin.—Tetanus antitoxin was administered intrathecally to 5 of the 25 most severely ill patients and 3 of these 5 survived, whereas in the same group only 1 patient of the 20 given antitoxin intravenously or intramuscularly recovered. This evidence supports the contention that tetanus antitoxin injected into the subarachnoid space is more effective in the treatment of human beings with severe tetanus than an equivalent amount administered by other routes. However, the superiority of this route of administration of antitoxin is not so definite when one studies the distribution of the patients who recovered as shown in figure 2. Dietrich [6] felt that many patients with tetanus, some of them with mild infections, died much sooner after being given antitoxin intrathecally or intravenously than they could have from untreated tetanus. Our cases support this point of view as regards tetanus antitoxin intrathecally. Patient E. B. illustrates that tetanus severe enough to have caused convulsions that produced fractures of four dorsal vertebrae can be survived without specific therapy, since his symptoms were subsiding by the time antitoxin was given:

E. B. (order number 33 in figure 2), a boy aged 6½ years, developed stiffness of the muscles of his neck and face eleven days before admission. Throughout the next six days there were increasing trismus and more generalized muscle spasms. On each of the last four days preceding admission a generalized convulsion occurred. There had been no fever, nor had any specific treatment been given. When admitted to the hospital on the eleventh day of his illness the patient displayed trismus, risus sardonicus, generalized increased muscle tone and a mid-dorsal kyphosis. Although sudden stimuli produced painful generalized spasms, 0.8 Gm. of sodium amytal in divided doses each day produced satisfactory relaxation during the first few hospital days, and no sedation was required thereafter. Following negative tests for sensitivity, 12,000 units of tetanus antitoxin was injected intramuscularly on the day of admission. Roentgen examination of the spine revealed compression of the bodies of the fourth, sixth and seventh dorsal vertebrae and fragmentation of the body of the fifth dorsal vertebra. The symptoms of tetanus, which were subsiding by the time of admission, gradually disappeared, and the compression fractures of his vertebral bodies responded satisfactorily to treatment.

Of the group classified as having mild infections, only 2 patients died. These were 2 boys (order numbers 35 and 56) who were given tetanus antitoxin intrathecally. Therefore it would seem that antitoxin intrathecally was chiefly responsible for these deaths. The case report of 1 of these 2 is as follows:

G. R. (order number 56 in figure 2), a boy aged 11 years, was admitted to the hospital because for the past three days he had had a right torticollis, inability to open his mouth more than 2 cm. and pain in his right cheek. Except for slight retraction of his head backward and to the right, physical examination revealed no additional findings. There was no irritability or spasm of any other muscles, no dysphagia, nor any additional neurologic signs. The consensus was that the most probable diagnosis was mild tetanus, and he was treated according to the usual method of treatment in use at that time. Intradermal tests for sensitivity to horse serum were negative. Under ether anesthesia clear spinal fluid was removed by lumbar puncture and a smaller volume of tetanus antitoxin, equivalent to 10,000 units, was allowed to flow slowly into the subarachnoid space. At the same time 10,000 units of antitoxin was given intravenously. His temperature rose rapidly from 100 F. before the serum administration to 106 F.

TABLE 1.—*Results of Treatment*

Route of Administration	Severe + Moderate Cases Order Numbers 1-31		Mild Cases Order Numbers 32-56	
	Cases	Deaths	Cases	Deaths
Intramuscularly only	14	11	11	0
Intravenously plus intramuscularly	10	9	10	0
Intrathecally plus intravenously and intramuscularly	7	2	4	2

four hours later. The patient became restless, but with no generalized spasms characteristic of tetanus. About ten hours after the serum had been given, the patient had a convulsion, then relaxed, had irregular respirations and died of respiratory failure.

Table 1 summarizes the results of therapy according to the route of administration of tetanus antitoxin for patients divided into two groups: severe plus moderate infections and mild infections. Table 1 illustrates the apparent superiority of the administration of tetanus antitoxin intrathecally, but of greater importance is its clear exposition of the serious nature of the reactions which may result from this type of treatment.

Type of Sedation.—Various types of sedation were employed in the treatment of the patients in this series. In general, solution of tribromoethanol and various barbiturates were employed, and no definite correlation could be made between the type of sedation and the final result. The sedation used for A. F. and C. R., who survived severe infections, is outlined in their case reports:

A. F. (order number 6 in figure 2), a girl aged 3 years, had an infected laceration of the scalp. The incubation period was seven days. Within twelve hours after the first symptoms a severe generalized convulsion occurred. The patient was sent to the hospital because of three more convulsions in rapid succession. At the time of admission she was having a convulsion which required ether anesthesia to control. Tetanus antitoxin 10,500 units was injected slowly into the lumbar subarachnoid space, an equal amount was given intravenously and 7,500 units was injected around the wound. For one hour

4. Spaeth, R.: Serum Therapy of Tetanus, with Special Reference to the Course of Antitoxin Titers in the Blood After Treatment with Specific Serum, Am. J. Dis. Child. **61**: 1146 (June) 1941.

5. Vener, H. I., and Bower, A. G.: Clinical Tetanus: Treatment in 100 Consecutive Cases with a Net Mortality Rate of 19 per Cent, J. A. M. A. **116**: 1627 (April 12) 1941.

6. Dietrich, H. F.: Tetanus in Childhood, with Special Reference to Treatment, Am. J. Dis. Child. **59**: 693 (April) 1940.

after these procedures the patient was in poor condition as the result of vasomotor collapse. During each of the first eight hospital days she was given from 6.5 to 15 Gm. of sodium bromide and from 50 to 100 cc. of a 25 per cent solution of magnesium sulfate intramuscularly. In spite of this sedation two to three convulsions occurred each day. During this period an additional 32,500 units of antitoxin was administered intramuscularly in divided doses. Lumbar punctures were done one to three times daily. Serum sickness, with temperatures reaching 106 F., was present on the ninth, tenth and eleventh days. A few days later no sedation was required. The patient was discharged on the forty-third day.

C. R. (order number 6 in figure 2), a boy aged 2½ years and weighing 12.3 Kg., developed dysphagia, trismus and stiffness of the muscles in his back and the right side of his neck although there was no history of an injury. The following day he was brought to the hospital, where physical examination revealed inability to open the mouth more than 1 cm., spasm of all muscle groups, hyperactive reflexes, enlarged cervical lymph nodes and a nearly healed abrasion over the right knee. Shortly after admission, within thirty-six hours of the onset of symptoms, the patient began to have severe generalized spasms accompanied by cyanosis in spite of sedation. After tests for sensitivity were negative, 10,000 units of tetanus antitoxin was given intravenously and 10,000 units intramuscularly. On the seventh hospital day another 10,000 units of antitoxin was administered intramuscularly. During the first thirteen days repeated doses of solution of tribromoethanol were used. A total of 1,400 mg. per kilogram of body weight was given during the first four days and 1,070 mg. per kilogram of body weight during the rest of the period. Because freedom from spasms could not be obtained through the use of solution of tribromoethanol alone, a 25 per cent solution of magnesium sulfate was injected intramuscularly repeatedly during the period from the fourth to the thirteenth day. The maximum amount given during a twenty-four hour interval was 100 cc. and the total amount used was 420 cc. The patient was discharged after fifty-two days in the hospital.

TABLE 2.—*Injuries Resulting in Tetanus*

Group	Injuries	Percentage of All Patients
A	5 Puncture wounds 3 Lacerations 2 Compound fractures 1 Crushed finger	20
B	3 Cuts on foot 3 Abrasions 2 Splinters in thigh 2 Blisters on foot 1 Minor burn	20
C	16 No injury located 7 Following vaccinations 5 Minor cuts on hand 5 Omphalitis 1 Boil on leg	60

Surgical Treatment.—In these 56 patients surgical débridement of the site of the initial lesion was carried out on 9 patients classified as having severe infections without influencing the fatal course of the disease and on 3 patients with mild infections, all of whom recovered. The futility of surgical excision of the site of experimental tetanus infections in animals, once symptoms have appeared, has been conclusively demonstrated by Abel and his co-workers.[7] Adequate amounts of antitoxin alone prevent the spread of tetanus toxin from its site of elaboration. This fact is manifested by the efficacy of prophylaxis by means of passive immunization. Hence there is no evidence that surgical intervention is indicated for a patient with clinical tetanus merely because the lesion may be the site of elaboration of the toxin.

This does not mean that proper surgical care of injuries, irrespective of the presence of tetanus, is not essential when the nature of the injury is such as to require operative intervention. Likewise it is to be stressed that any injury which might result in tetanus should be given adequate surgical treatment at the time the injured person is first seen by a physician.

THE PREVENTION OF TETANUS

The methods of preventing tetanus are the proper immediate surgical treatment of injuries, passive immunization of injured patients with tetanus antitoxin and active immunization against tetanus. The latter may be accomplished by means of an initial series of injections, to be followed by "booster" doses of toxoid at the time of injury or by means of toxoid injections so spaced as to maintain adequate protection continuously. A consideration of the types of injuries constituting the probable ports of entry for tetanus spores in these 56 cases enables one to evaluate the methods of preventing tetanus.

The presumed ports of entry are given in table 2.

Group A, constituting 20 per cent of all the cases, consisted of injuries that usually would be treated by a physician and that were of such a nature that many physicians would feel it essential to give passive immunization with tetanus antitoxin or a "booster" injection of tetanus toxoid to patients previously actively immunized. Hence, theoretically, these 11 cases could have been prevented by active or passive immunization. In the present series only 1 patient (order number 11 in figure 2) was given passive immunization. The day after she sustained a compound fracture of her arm she was given 1,500 units of tetanus antitoxin, an amount generally conceded to be inadequate when an injury with much devitalized tissue is present.

Group B, constituting 20 per cent of the cases, represented injuries for which a physician was often not consulted and which are so infrequently accompanied by any sequelae that it is unlikely that passive immunization would be used. A physician might well use a "booster" dose of toxoid if the patient had been previously actively immunized. Hence, reliance on the use of passive immunization would probably not have prevented any of these 11 cases. It is doubtful if reliance on the method requiring the use of "booster" doses of toxoid would have prevented all of them, because medical advice was so seldom sought.

Group C, containing 60 per cent of the cases, presented injuries from which tetanus could not have been prevented by reliance on either passive immunization or "booster" doses of toxoid, because either the injury was unknown or was of such a nature that these methods of preventing tetanus would not ordinarily be utilized.

Therefore it is evident that tetanus will be most satisfactorily controlled by a program for active immunization which will produce and constantly maintain a high degree of immunity. An additional dose of toxoid should be given whenever an injury particularly prone to result in tetanus is sustained, because, at the present time, insufficient evidence is available to determine whether or not such a measure can be safely omitted, although infection with tetanus organisms probably would act as a "booster" dose. The most practical

7. Abel, J. J., and Hampil, B.: Researches on Tetanus: Some Historical Notes on Tetanus and Commentaries Thereon, Bull. Johns Hopkins Hosp. **57**: 343 (Dec.) 1935.

method of active immunization to maintain a high degree of immunity against tetanus is still to be determined, but the studies of Miller and Saito [8] and of Peshkin [9] indicate methods by which serologically adequate immuity can be achieved.

It is especially important to be sure that any patient who is given tetanus antitoxin is actively immunized subsequently. Several of the patients reviewed had received prophylactic tetanus antitoxin months or years previously, and the resulting sensitivity made serum therapy more difficult and increased the risk of serious reactions. The availability of serums prepared in other animals besides the horse has not entirely eliminated this problem.

SUMMARY AND CONCLUSIONS

A method for determining the relative severity of each infection in a series of patients with tetanus was adopted. Arrangement of 56 children with tetanus in order of the severity of the infection assisted in evaluating the various methods of treatment employed:

1. There was no evidence that administration of tetanus antitoxin in amounts up to 80,000 units was more effective than amounts of 30,000 units.

2. The administration of tetanus antitoxin intrathecally was suggestively, but not definitely, more effective than giving the same amount of antitoxin by the intramuscular or intravenous routes.

3. Fatal reactions occurred from tetanus antitoxin given intrathecally to some patients who in all likelihood would have survived their tetanus infections. Hence tetanus antitoxin should not be used intrathecally in any but the most severe cases, if it should be used at all, until a preparation is available which is incapable of producing severe reactions.

4. Excision of the site of infection, once clinical symptoms of tetanus were present, did not ameliorate the course of the disease.

That further reduction in the incidence of tetanus can be accomplished only by producing and constantly maintaining a high degree of active immunity against this infection was disclosed by a study of the initial lesions in these 56 patients.

8. Miller, J. J., Jr., and Saito, T. M.: Concurrent Immunization Against Tetanus, Diphtheria and Pertussis: A Comparison of Fluid and Alum Precipitated Toxoids, J. Pediat. **21**: 31 (July) 1942.

9. Peshkin, M. M.: Immunity to Tetanus Induced by a Third Dose of Toxoid Four Years After Basic Immunization, Based on a Study of 25 Allergic Children, Am. J. Dis. Child. **69**: 83 (Feb.) 1945.

War Neuroses.—One of the most striking phenomena seen in the conversion states, and one almost defying description, is the weird, wobbly, disorganized gait of astasia-abasia, with which so many of these patients are affected. The patient may be capable of perfectly normal movements in the sitting or the horizontal position, even though many of them display constant gross tremors of the legs only with great difficulty. The legs wobble about in every direction; the trunk is agitated by a series of uneven jerks. Time and again the patient seems on the point of losing his balance and toppling over, yet this seldom occurs. At other times the knees seem to give way altogether, causing the patient to sink to the floor. If the patient attempts to stand still, his body bobs up and down continuously. This condition is accompanied by little or no anxiety. The patient struggles persistently to control the wanton movements. He regards the disability as a disease of the nerves and muscles of the legs and does not see the actual relation between his symptoms and his war experience. In some cases the symptom appears as a transitory phase in the anxiety states.—Grinker, Roy R., and Spiegel, John P.: War Neuroses, Philadelphia, Blakiston Company, 1945.

THE TREATMENT OF EARLY SYPHILIS WITH PENICILLIN

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The effectiveness of penicillin in the treatment of early syphilis in human beings was first recorded, in a preliminary report, by Mahoney, Arnold and Harris [1] in December 1943. Since then a number of publications [2] also of preliminary nature have appeared. It is my purpose in the present communication to detail the results of treatment of 96 patients, more than 70 per cent of whom have been observed for a year or more following treatment. These patients were treated between Nov. 17, 1943 and May 18, 1944.

MATERIAL AND PROCEDURE

The series consisted of 92 men and 4 women, 29 of whom were white and 67 Negro patients. They varied in age from 18 to 38 years, approximately two thirds being younger than 25.

Lesions of primary or secondary syphilis were present in every case, and the diagnosis was established in 89 by the demonstration of Treponema pallidum in the lesions, and in the other 7 by repeatedly positive serologic tests for syphilis. The approximate duration of the lesions was five to three hundred days, although in some 60 per cent of the patients it was less than thirty days. When treatment was initiated 19 patients were in the seronegative primary stage, 34 in the seropositive primary stage and 43 in the secondary stage. Two patients in the secondary stage had an additional complication, 1 syphilitic iritis, the other syphilitic periostitis of the tibias. Besides early syphilis, 2 men had acute gonococcic urethritis and a third had suppurative inguinal lymphogranuloma venereum.

All patients were hospitalized and treated identically: they were given sixty intramuscular injections of 20,000 units of penicillin in 1 cc. of saline solution at three hour intervals, day and night, for seven and one-half days, a total of 1,200,000 units. Seventeen different lots of sodium penicillin, supplied by two manufacturers, were employed.

A titered Kahn test and a Wassermann test of the blood were performed immediately before and after treatment, and these tests were repeated at monthly intervals, or more often, during the period of observation. The cerebrospinal fluid was examined before treatment in every patient and reexamined in most of them six months or more after treatment. Complete blood counts, urine analyses, estimations of nonprotein nitrogen or urea of the blood, icterus indexes and sedimentation rates were made before and after treatment in a large number of the patients.

REACTIONS TO PENICILLIN

A Herxheimer reaction occurred in 88 (91.6 per cent) patients during the first day of treatment. In 65 it consisted of fever, chill or chilly sensations, headache

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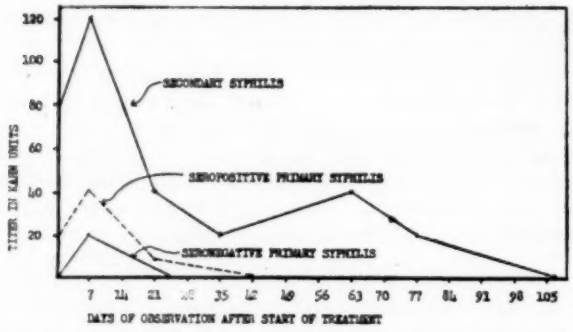
and malaise. In 1 patient the temperature rose to a height of 105.2 F. but in most it was between 101 and 103 F. The temperature was practically always normal after twenty-four hours in spite of the uninterrupted administration of penicillin. Besides the febrile reaction 21 patients had a temporary exacerbation of the cuta-



Provocative effect of treatment on the titered serologic test and the pattern of decline to a negative reaction in 3 typical cases.

neous and mucosal lesions, evidenced by increased swelling and redness and frequently by pain in the chancre and the enlarged regional lymph nodes. Two patients had exaggeration of the cutaneous lesions without a febrile reaction.

A number of minor reactions were observed after the first day of treatment. Several patients complained of mild abdominal cramps and nausea, of brief duration, and 2 patients vomited twice. Two patients had a secondary rise of temperature to 101 F. on several days during treatment. Two patients had mild urticaria lasting less than forty-eight hours; 2 had generalized pruritus without eruption; 1 had a papular multiform erythema, distributed over the elbows, forearms, knees and legs, that lasted about two weeks; 3 had herpes labialis and 2 herpes progenitalis following a febrile Herxheimer reaction.

No significant abnormalities of the blood counts, the nonprotein nitrogen, the blood urea or the icterus index were noted. The sedimentation rate was moderately prolonged after completion of treatment in 19 patients; the reason for this was not apparent.

TABLE 1.—*Relapses Classified According to Stage of Disease, Type of Relapse and Time of Appearance*

Case No.	Stage of Disease	Type of Relapse	Number of Days After Treatment That Relapse Appeared
12	Secondary	Serologic with abnormal CSF *	123
21	Secondary.	Cutaneous	116
24	Secondary	Neurologic with abnormal CSF *	105
25	Secondary	Neurologic with abnormal CSF *	123
70	Secondary	Mucocutaneous	103
88	Secondary	Mucocutaneous	243
97	Secondary	Mucocutaneous	105
69	Seronegative Primary	Mucocutaneous	401

* Cerebrospinal fluid.

It is important to note that it was not necessary to interrupt or suspend treatment in a single patient on account of reaction.

RESULTS

Early Effects of Treatment.—Dark field examinations were repeated at three hour intervals after the onset of therapy in 26 patients with suitable lesions. Treponemes could not be demonstrated after periods of three to fifteen hours, and in a majority of instances after nine hours.

Chancres and mucous patches healed and macular eruptions disappeared usually by the time treatment was finished. However, exceptionally large and deeply ulcerated chancres and heavily infiltrated papular eruptions required a longer time for complete healing. In every case the lesions healed without additional treatment, and no evidence of treatment resistance was encountered. The patient with syphilitic iritis and the one with syphilitic periostitis of the tibias both responded rapidly and completely to treatment.

Treatment often had a provocative effect on the serologic tests for syphilis. Immediately after the termination of treatment, 9 of the 19 seronegative primary cases showed a positive serologic test for syphilis, and most of the seropositive primary and secondary cases showed an increase in the titer of the Kahn test.

Reversal of the serologic tests to negative was the end result of a gradual decline in the titer of the quantitative test during the post-treatment period. The accompanying chart demonstrates the provocative effect of treatment on the serologic tests and also the pattern of serologic decline to negativity in typical cases of seronegative primary, seropositive primary and secondary syphilis. In general, the speed of seroreversal depended on the initial titer of the Kahn test; that is,

TABLE 2.—*Blood Serologic Response and Relapses in Seronegative Primary Syphilis*

Period of Observation, Months	Number of Cases	Serologic Tests for Syphilis			Relapses
		Negative	Doubtful	Positive	
0 - 6	0	..	..	..	..
6 - 12	4	4	0	0	0
12 - 18	11	10	0	0	1
Over 18	4	4	0	0	0
Total	19	18	0	0	1

cases with a high initial titer required a longer time to achieve seronegativity than cases with a low initial titer. In the majority, complete reversal of the tests took place within four months following treatment. There were a number of exceptions, especially in secondary syphilis, with high initial titers, in which fixed seronegativity was attained only after a lapse of six to twelve months. In these cases the serologic reactions fluctuated in a narrow zone between negative, doubtful and positive reactions of low titer before a stationary negative state was maintained.

The 2 men who also had acute gonococcic urethritis obtained a complete cure of this infection. There was no apparent therapeutic effect on the lymphadenitis of lymphogranuloma venereum. The lymph nodes suppurated and discharged purulent material and did not heal until the sulfonamide drugs were administered.

RELAPSES

In table 1 are presented the details of 8 cases classified as relapses. Seven occurred in patients with secondary syphilis, and 1 in a patient with seronegative primary syphilis. In 5 patients there was a reappearance of lesions of the skin or mucous membranes, and in 4 of these treponemes were demonstrated in the lesions. All 5 patients also had a concomitant serologic relapse. Another patient suffered only a serologic relapse, but reexamination of his cerebrospinal fluid, which had been normal prior to treatment, showed it to be abnormal. The 2 other patients had a neurologic relapse, accom-

panied by serologic relapse and abnormal cerebrospinal fluid; 1 of these men had acute syphilitic meningitis, the other a monoplegia, and both had had normal cerebrospinal fluid before treatment.

In 6 patients the relapse was noted approximately 103 to 123 days after the inception of treatment, that is, in the fourth month of observation. These included the 2 patients with neurologic relapse, the 1 with serologic relapse and abnormal spinal fluid, and 3 with cutaneous or mucocutaneous relapse. In none of these 6 cases could any serious consideration be given to the question of reinfection, but the other 2 cases (88 and 69) are controversial in this respect and merit special attention.

TABLE 3.—*Blood Serologic Response and Relapses in Seropositive Primary Syphilis*

Period of Observation, Months	Number of Cases	Serologic Tests for Syphilis			Relapses
		Negative	Doubtful	Positive	
0 - 6	3	2	0	1	0
6 - 12	1	1	0	0	0
12 - 18	24	23	1	0	0
Over 18	6	6	0	0	0
Total	34	32	1	1	0

In case 88 negative serologic tests for syphilis occurred at 106 days after treatment and again at 120, 151, 181 and 213 days. At 207 days the cerebrospinal fluid was normal. At 243 days the titer of the Kahn test rose to 200 units and the patient was returned from another station for study. He had had an attack of acute gonococcic urethritis about two months previously, that is at about 183 days after treatment, and at that time the serologic tests were negative. The urethritis had responded to treatment with the sulfonamide drugs. Examination revealed a generalized lymphadenopathy and a papule of the scrotum in which Treponema pallidum was present. No evidence of a new chancre or of activity at the site of the former chancre was observed.

Patient 69 remained seronegative throughout treatment and up until 123 days following treatment, at which time he was lost from observation because of discharge from the army. Information was later received that this man had been admitted to a civilian hospital on May 19, 1945, about thirteen months after treatment, because of the presence of several penile erosions in which treponemes were demonstrated. On May 21, 1945 the Kahn test of the blood was positive (20 units) and the cerebrospinal fluid was normal. No exact information was obtainable as to the location of the new lesions in relation to the original chancre.

TABLE 4.—*Blood Serologic Response and Relapses in Secondary Syphilis*

Period of Observation, Months	Number of Cases	Serologic Tests for Syphilis			Relapses
		Negative	Doubtful	Positive	
0 - 6	9	0	2	1	6
6 - 12	7	5	0	1	1
12 - 18	20	20	0	0	0
Over 18	7	7	0	0	0
Total	43	32	2	2	7

LATE EFFECTS OF TREATMENT

Table 2 presents the results in 19 patients with seronegative primary syphilis. Of these, 18 were seronegative when last examined, 2 between six and nine months after treatment, 2 between nine and twelve months, 5 between twelve and fifteen months, 5 between fifteen and eighteen months, and 4 between eighteen and twenty-one months. One case (69) was classified as a relapse, and the facts have been detailed under the subject of relapses.

Table 3 presents the results in 34 patients with seropositive primary syphilis. The last serologic test was negative in 32 of these patients, at the following intervals after treatment: 1 in less than three months, 1 between three and six months, 1 between nine and twelve months, 14 between twelve and fifteen months, nine between fifteen and eighteen months and 6 between eighteen and twenty-one months. One patient had a doubtful Kahn test between twelve and fifteen months. One patient had a positive Kahn test, with a titer of 20 units as compared with a pretreatment titer of 200 units, between three and six months after treatment. No relapses have thus far been observed in this group.

Table 4 presents the results in 43 patients with secondary syphilis. The last serologic test was negative in 32 patients at the following intervals after treatment: 4 between six and nine months, 1 between nine and twelve months, 6 between twelve and fifteen months, 14 between fifteen and eighteen months, and 7 between eighteen and twenty-one months. Two patients had doubtful Kahn tests when last seen, 1 in less than three months and 1 between three and six months. A positive Kahn test was present in 1 patient between three and six months and in another between six and nine months, but in both cases the titer was considerably lower than that found prior to treatment. Seven relapses were observed in this group, and these have been explained in the preceding section under relapses.

TABLE 5.—*Summation of Blood Serologic Response and Relapses in Entire Series of 96 Cases*

Period of Observation, Months	Number of Cases	Serologic Tests for Syphilis			Relapses
		Negative	Doubtful	Positive	
0 - 6	12	2	2	2	6
6 - 12	12	10	0	1	1
12 - 18	55	53	1	0	1
Over 18	17	17	0	0	0
Total	96	82	3	3	8

TABLE 6.—*Results of Cerebrospinal Fluid Examinations*

Stage of Disease	Number of Cases	Pretreatment Fluid		Post-Treatment Fluid		Not Examined
		Normal	Abnormal	Normal	Abnormal	
Seronegative primary	19	19	0	18	0	1
Seropositive primary	34	30	4	31	0	3
Secondary	43	36	7	37	3	3
Total	96	85	11*	86	3†	7

* Of the 11 abnormal fluids, 8 were normal on reexamination and 3 have not yet been reexamined.
† The 3 abnormal fluids were found in patients who suffered a clinical or serologic relapse and whose fluid was normal prior to treatment.

Table 5 is a summation of the therapeutic results in the entire series. Twelve patients were observed less than six months, and of these 6 relapsed; of the other 6, 2 were seronegative, 2 seropositive and 2 had a doubtful Kahn test when last seen. Twelve more patients were observed between six and twelve months; in this group there was 1 relapse, and 10 were seronegative and 1 seropositive at the last examination. Among the 72 patients observed longer than twelve months there was 1 relapse; 70 were seronegative and 1 had a doubtful Kahn test when last examined.

CEREBROSPINAL FLUID EXAMINATIONS

Eleven of the 96 patients had an abnormal spinal fluid prior to treatment, as shown in table 6. In 6 patients the abnormality consisted solely of an increased cell count, between 9 and 14 cells per cubic millimeter, and in the other 5 there was a positive complement fixation reaction. Eight of these patients (5 with pleocytosis and 3 with positive complement fixation reaction) had a normal cerebrospinal fluid between 179 and 472 days after the onset of treatment. The other 3 patients have not as yet had a reexamination of the fluid.

The only abnormal cerebrospinal fluids among the 89 examined subsequent to treatment were found in 3 patients who suffered a relapse in the fourth month of observation; these have already been described in the section on relapses.

COMMENT

In both intensive penicillin therapy [3] and intensive arsenotherapy [4] it has been shown that immediate satisfactory response, as indicated by disappearance of treponemes, healing of lesions and reversal of serologic tests, is no criterion of ultimate satisfactory outcome. Grossly inadequate doses of the treponemicides produce immediate results similar to those obtained with therapeutically adequate doses. The effectiveness of a given treatment schedule must be judged by the incidence of relapse, which is greatest in the first six months after treatment, and by prolonged observation of patients to make certain that early satisfactory progress is maintained permanently.

Six of the eight relapses in the present series occurred in the fourth month of observation and were undeniably relapses rather than reinfections. Patient 88 (secondary syphilis) relapsed about eight months after treatment, and patient 69 (seronegative primary syphilis) about thirteen months after treatment. Both these men admitted frequent opportunity for reinfection, both had been seronegative for some time, and both had a normal cerebrospinal fluid on reexamination. Since the rigid criteria for reinfection could not be met, these cases are considered relapses in the present discussion. It follows, then, that the incidence of observed relapse in the present series of 96 patients was 8.3 per cent. Furthermore, it is significant that the incidence of relapse, by stage of disease, was as follows: in seronegative primary syphilis, only 1 (5.3 per cent) of 19 patients; in seropositive primary syphilis, none of 34 patients; and, in secondary syphilis, 7 (16.3 per cent) of 43 patients.

The patients followed for a period longer than twelve months and up to twenty months present a most reassuring serologic and clinical picture. Among 72 such patients (table 5) 70 were seronegative and 1 had a doubtful Kahn test when last examined. Of the eight relapses in the entire series, only 1 occurred after twelve months, and this patient (69) may have had a reinfection. The cerebrospinal fluid was normal in all 72 patients after treatment. Large accumulated experience with intensive arsenotherapy has revealed not only that the highest incidence of relapse occurs in the first six months following treatment, but that patients who are clinically and serologically well after twelve months of observation seldom suffer a relapse at a later period. Since the pattern of therapeutic response to intensive penicillin therapy in early syphilis is practically identical with that of intensive arsenotherapy, it is probable that the 71 patients in this series who are progressing satisfactorily after twelve months of observation will continue to do so.

The beneficial effect of the treatment on abnormal cerebrospinal fluid is shown in table 6. The only abnormal fluids among the 89 reexamined were found in 3 patients who developed a serologic or clinical relapse in the fourth month of observation. Furthermore, in 8 patients with abnormal fluids prior to treatment all were normal when examined six months or more after treatment.

The incidence of observed relapse, 1 in 53 patients with seronegative and seropositive primary syphilis, and 7 in 43 patients with secondary syphilis, suggests that the treatment was adequate for primary syphilis but inadequate for secondary syphilis. Experiences with smaller doses of penicillin [3] and with intensive arsenotherapy [4] have been similar in this respect; a higher failure rate was generally found in the secondary stage of syphilis than in the primary stage. It is possible that the results in secondary syphilis can be improved by increasing the size of the individual dose of penicillin, by lengthening the course of treatment or by both. Certain unknown factors, such as the in vivo sensitivity of Treponema pallidum and the exact nature of the increased resistance to treatment of secondary syphilis, make it impossible to predict which of these possibilities is most likely to increase the percentage of cures. It is known, for example, that the height of the blood level can be doubled by doubling the size of the individual dose; [5] however, this may be of no great advantage since doubling the blood level increases the duration of penicillin in the blood by only about one third. On the other hand, it may be that a higher level will give greater penetration into relatively inaccessible areas. It is obvious that the answer can be obtained only empirically—by treating large numbers of patients with different regimens.[6] As a departure the armed forces and certain civilian agencies are now following the exact treatment schedule used in the present study but are doubling the size of each individual dose; i. e., 2,400,000 units in sixty injections. It may be found necessary later to prolong the duration of therapy, although from the standpoint of time and expense this is naturally undesirable.

While the treatment of early syphilis by the schedule described is totally inapplicable to the outpatient clinic, it has two enormous virtues: it assures complete treatment of practically all, if not all, patients, a goal never even approached by the outpatient, arsenotherapeutic clinic, in which a high percentage of the patients became discouraged and stopped treatment before it was completed; and it does this without significant toxicity. Perhaps the desideratum of a practicable and successful ambulatory therapy for early syphilis will be met by the use of penicillin suspended in beeswax-peanut oil mixtures,[7] by administering one or two intramuscular or subcutaneous [8] injections daily for eight or ten consecutive days.

3. Moore, J. E.; Mahoney, J. F.; Schwartz, W. H.; Sternberg, T. H., and Wood, W. B.: The Treatment of Early Syphilis with Penicillin, J. A. M. A. **126**:67 (Sept. 9) 1944.

4. Leifer, W.; Chargin, L., and Hyman, H. T.: Massive Dose Arsenotherapy by Intravenous Drip Method, J. A. M. A. **117**: 1154 (Oct. 4) 1941.

5. Rammelkamp, C. H., and Keefer, C. W.: The Absorption, Excretion, and Distribution of Penicillin, J. Clin. Investigation **22**:425 (May) 1943.

6. A large scale investigation, with different time-dose plans, is now going on under the general auspices of the Committee on Medical Research of the Office of Scientific Research and Development and under the specific direction of the Subcommittee on Venereal Diseases, National Research Council. Moore, Mahoney, Schwartz, Sternberg and Wood.[3]

7. Romansky, M. J., and Rittman, G. E.: Penicillin: I. Prolonged Action in Beeswax-Peanut Oil Mixture; II. Single Injection Treatment of Gonorrhea, Bull. U. S. Army M. Dept., October 1944, No. 81, p. 43.

8. Kirby, W. M. M.; Leifer, W.; Martin, S. P.; Rammelkamp, C. H., and Kinsman, J. M.: Intramuscular and Subcutaneous Administration of Penicillin in Beeswax-Peanut Oil, to be published.

SUMMARY

1. Each of 96 patients with early syphilis received sixty intramuscular injections of 20,000 units of penicillin in saline solution at three hour intervals, day and night, for seven and one-half days.

2. A Herxheimer reaction occurred in 88 (91.6 per cent) of the patients, and also some minor cutaneous, gastrointestinal and febrile reactions: in no case was it necessary to suspend or interrupt treatment because of reactions.

3. A relapse was observed in 8 (8.3 per cent) of the 96 patients, but 2 of these may have been reinfection. Relapse occurred in 1 (5.3 per cent) of 19 seronegative primary cases, in none of 34 seropositive primary cases and in 7 (16.3 per cent) of 43 secondary cases.

4. Of 72 patients followed from twelve to twenty months, 70 were seronegative and 1 had a doubtful Kahn test of the blood at the last visit; the other patient relapsed in the thirteenth month, and he may have had a reinfection.

5. The cerebrospinal fluid of 89 patients was reexamined six months or later following treatment; it was normal in 86, including 8 with abnormal pretreatment fluids: the 3 abnormal fluids were coincidental with a clinical or serologic relapse three to four months after treatment.

6. Although the results of this study must be considered preliminary—and the optimum treatment schedule, particularly for secondary syphilis, is still to be worked out—the findings suggest that the eight day treatment of early syphilis with 1,200,000 units of penicillin does cure early syphilis.

AN EFFECTIVE METHOD FOR THE CONTROL OF TRICHINOSIS IN THE UNITED STATES

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INCIDENCE OF TRICHINOSIS IN THE UNITED STATES

A large number of recent surveys of necropsy material in various localities in the United States has shown an average incidence of 16 per cent of trichinous infection.[1] When the methods of study were more thorough, an incidence as high as 36 per cent has been found.[2] The amazingly high frequency of parasitism with Trichinella spiralis revealed by these surveys caused Hall[3] to emphasize that "the United States has the greatest problem of trichinosis of any country in the world."

It is now generally recognized that man acquires trichinosis primarily from eating trichinous pork and that the hog becomes infected primarily from eating scraps of uncooked trichinous pork present in garbage. The average incidence of trichinosis among hogs in the United States during the past fifty years has remained practically unchanged at a level of approximately 1.5 per cent. During 1944, 96,849,000 hogs were slaughtered in the United States and the total production of pork was 12,893,000,000 pounds.[4] It is estimated that this amount of pork would furnish about 30 billion individual servings, or approximately 200 servings per person in the United States. Since 1.5 per cent of the hogs are trichinous, 3 of these 200 servings of pork would contain trichinae. During 1944 most of the pork produced in this country was purchased by the armed forces and other governmental agencies for export. However, if it is assumed that in times of peace the annual per capita consumption of pork in the United States amounts to 200 servings, then each American consumes, on the average, 3 servings of trichinous pork each year; and since the average length of life of Americans is now over 64 years,[5] there will be consumed for each American during his lifetime nearly 200 meals of pork containing trichinae. If the ingested trichinae are nonviable or have been killed by proper cooking or other method of processing, no infection will result. But it must be remembered that it requires the ingestion of viable trichinae in raw, undercooked or inadequately processed pork from only one meal to produce infection.

MORBIDITY AND MORTALITY FROM TRICHINOSIS

While it is true that in the vast majority of instances of parasitism with trichinae the infection is subclinical, there is no doubt that many infections produce symptoms which are undiagnosed. Gastrointestinal symptoms often appear three to fourteen or more days after the ingestion of trichinous pork, but the cause of this enteritis is seldom diagnosed or even suspected unless the patient becomes so ill as to require hospitalization or the disease occurs simultaneously in a number of persons. Considering the large chance for most people in the United States of eating pork containing viable trichinae at least once during their lifetime, it is not surprising that at least 16 per cent of Americans are found at autopsy to have been infected with trichinae. The morbidity rate in the United States is not definitely known, but the mortality rate in cases of clinically recognized trichinosis is 5 to 6 per cent.

TRICHINOSIS UNCONTROLLED AT PRESENT

The United States is in much the same situation today as regards the control of trichinosis as it was thirty or more years ago with regard to the question of pasteurization of milk. Despite the opposition at that time to the general adoption of pasteurization, the introduction of this method ushered in a new era in dairy sanitation. While there is considerable expense connected with pasteurization of milk, particularly for equipment, buildings and personnel, no one will now deny that the expense incident to effective pasteurization is worth while and even necessary for the protection of human health. And the public gladly assumes this extra expense in order to be assured of milk that is safe for human consumption. Prior to the general acceptance of pasteurization, many people were loath to drink milk, which they knew was often filthy; but today, when nearly all milk is pasteurized and produced under more sanitary conditions, the prejudice against drinking milk has practically disappeared. The dairy industry has profited vastly from the many advantages that it has gained as a result of the adoption of pasteurization of milk.

From the Departments of Pathology, Wayne County General Hospital, Eloise, Mich., and Wayne University College of Medicine, Detroit.

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5. Longevity of the American People in 1943, Statist. Bull., Metropolitan Life Insurance Company **26**: 3-6, 1945.

On the other hand, during the past fifty years very little progress has been made in the control of trichinosis in the United States. Approximately 70 per cent of American pork is prepared in plants which are under governmental inspection and carries the stamp "U. S. inspected and passed." This label on nonprocessed pork does not indicate that the meat is free from living

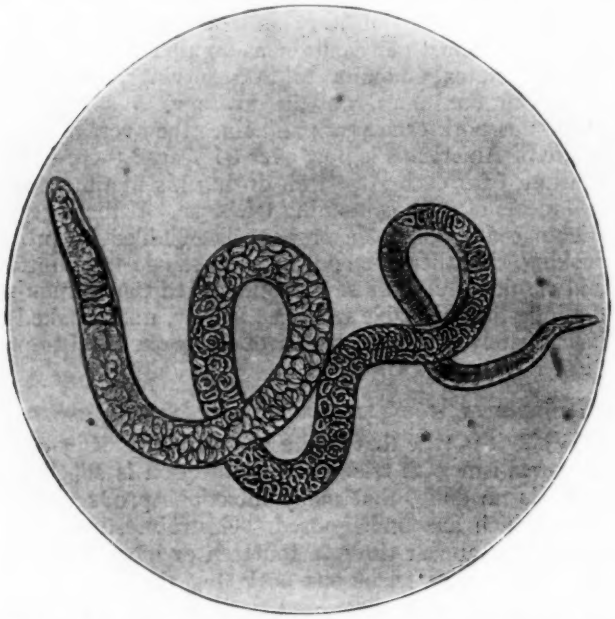

Fig. 1.—Adult gravid female trichina recovered from intestinal lumen of white rat six days after ingestion of trichinous meat. The female worm gives birth to from 1,000 to 1,500 trichina larvae. Slightly reduced from a photomicrograph with a magnification of 100 diameters.

trichinae. Meat-packing plants which are under governmental inspection are required to observe federal regulations for the processing of pork products of a kind ordinarily eaten without cooking, and such processed products may be expected to be free from viable trichinae. Other nonprocessed pork prepared in plants under federal inspection, and all pork prepared in plants not under federal inspection, may contain viable trichinae and may cause illness or death if consumed after inadequate cooking. The danger of contracting trichinosis from eating sausages containing pork is particularly great, since sausages are frequently eaten raw or insufficiently cooked.

Since only about 70 per cent of the pork is produced in plants under federal inspection and since in such plants it is only the processed products that may be expected to be free from viable trichinae, the larger part of the total American pork supply which reaches the kitchen is open to the danger of carrying live parasites. The consumer assumes that the pork that he eats is a safe food, but he is seldom aware that it is actually free from living trichinae. Furthermore, the consumer may knowingly or unknowingly eat pork that has been placed in certain meat products, such as frankfurters, hamburger, sausages, meat loaf and chop suey, which he purchases either in the butcher shop or the delicatessen store or in the restaurant, and if such meat products have been insufficiently cooked or inadequately processed he may run the risk of acquiring trichinosis. The question may properly be asked, "Why should the consumer of pork constantly be exposed to the danger to health and life from trichinosis?" Surely the public will be grateful for any method which will adequately safeguard its health in the consumption of this important food, and it will gladly assume the small extra expense incurred in the production of a safe supply of pork.

In recent years numerous legal suits have been instituted because of illness or death resulting from trichinosis. The claims have been based, in most of these instances, on a breach of implied warranty of the fitness of the pork as a food for human consumption and in many cases on negligence in the methods of production of pork as a food. Most of the claims have been brought against the packer, and in the majority of suits the judgment has been rendered in favor of the plaintiff. In a number of instances the awards granted have been remarkably high.

The adoption of a method which will produce a supply of pork free from viable trichinae will remove the objection which many people now have to eating pork because of the danger of contracting trichinosis. After a method of control of trichinosis has been adopted, it is probable that other measures will be instituted for the improvement of sanitary conditions in the swine industry, so that the public will be less inclined to associate conditions of filth with the raising of hogs. From a purely business point of view, the swine raisers and the pork packers will surely benefit greatly from utilizing a satisfactory means for the control of trichinosis.

It is logical and inevitable that sooner or later a method will be adopted for producing a supply of pork that is free from the danger of causing trichinosis in man. The prevention of trichinosis in man consists essentially in the elimination of the disease from hogs. The high average incidence of approximately 1.5 per cent of trichinosis in hogs in the United States, which has remained unchanged during the past fifty years, stands in sharp contrast to an incidence of trichinous infection in hogs in Canada of 0.57 per cent, in Poland

Fig. 2.—Portions of gravid female adult trichina embedded in mucosa of duodenum of white rat six days after ingestion of trichinous meat. The mother worm is depositing her larvae at this point, from which they will eventually reach the skeletal muscle and encyst in it. Reduced from a photomicrograph with a magnification of 100 diameters.

of 0.05 per cent, in Germany of 0.001 per cent and in Copenhagen of only 0.00075 per cent. Official statistics are available covering the microscopic examination for trichinae in 8,000,000 hogs in the United States from 1898 to 1906 and in 6,000,000 hogs in Copenhagen from 1910 to 1935. These statistics show that in the United

States over 14,000 hogs per million were found trichinous, compared to only 7 hogs per million in Copenhagen.

AVAILABLE CONTROL MEASURES

Measures which have been advocated in the control of trichinosis include (1) destruction of rats, (2) elimination of garbage and of offal from the feed of hogs,

Fig. 3.—Encysting larvae in human muscle. Infection about 7 weeks of age. Note intense inflammatory reaction. Reduced from a photomicrograph with a magnification of 75 diameters.

(3) cooking of garbage which is to be fed to hogs, (4) microscopic inspection of pork, (5) proper processing of pork products which are customarily eaten without cooking and (6) education of the public in the necessity of adequate cooking of pork and pork products which are customarily cooked before being eaten. Skin-testing of hogs with a trichina antigen prior to slaughter, as a possible method of control,[6] has been found to be unreliable in detecting animals which harbor trichinae.

While rats are frequently infected with trichinae, it is generally agreed that it is a very rare occurrence for a pig to eat a rat and therefore for a pig to acquire trichinosis from a rat. It is, of course, highly desirable from the point of view of general sanitation that methods be instituted for the extermination of rats, but it must be admitted that the rat plays a minor role in the spread of trichinosis to the pig and thence to man.

Education of the public through the medium of the newspapers, by use of posters displayed in butcher shops and particularly through instruction in the public schools should not be neglected, but such education in itself will not eradicate trichinosis in man or pig.

There are three principal methods of control of trichinosis: (*a*) microscopic inspection of pork, (*b*) cooking of all garbage fed to hogs and (*c*) processing of all pork prior to its ultimate sale.

MICROSCOPIC INSPECTION

The microscopic inspection of pork has been practiced in a number of countries and has proved to be quite effective. This is the method which has been used most extensively in various parts of Germany since 1865. During the years 1883 to 1924 in the city of Berlin 4,337 hogs were found to be trichinous, and during these forty-one years no case of human trichinosis was reported in that city from eating pork that passed inspection. In other parts of Germany trichinosis from eating raw pork which had passed microscopic inspection has been reported sporadically, and yet there can be no question but that in Germany the systematic enforcement of this method has almost eradicated the disease. Other countries which require microscopic inspection of pork for trichinae include Denmark, Sweden, France, Argentina and Chile. The United States also had compulsory microscopic inspection of pork during the period from 1891 to 1906, but only for pork that was intended for export. This inspection was authorized by an act of Congress and was necessitated by the virtual exclusion of American pork from the European market, an embargo which was highlighted by specific decrees issued by Bismarck in 1880 and 1883 which prohibited the importation of American pork into Germany. Despite the method of microscopic inspection which was instituted in the United States in 1891, Germany in 1892 required the reinspection of American pork and in 1898 refused altogether to accept the certification of inspection of the United States Department of Agriculture. Consequently in 1906 Congress adopted a new measure which no longer required that pork be inspected microscopically.

Microscopic inspection has therefore never been applied in the United States to pork consumed in this country. A theoretical objection has been raised that this method is not practical, since it is too time consuming for present high-speed American requirements. Microscopic inspection has recently been introduced into Chile, where the use of the phototrichinoscope has been found to expedite the examination. By this method a moving trichinoscope or compressor containing several specimens of meat from each hog is projected onto a screen, where it is scrutinized by the inspector for the possible presence of trichinae. If the method of microscopic inspection should be instituted in the United States for the control of trichinosis, it is safe to assume that suitable devices for a sufficiently rapid examination of the meat would soon be developed.

The method has also been criticized as being too expensive. It is true that the expense of the microscopic inspection would undoubtedly be greater than either of the other methods of control under consideration, since it would require, in addition to buildings and apparatus, a vast corps of inspectors. The relatively greater cost of microscopic inspection, therefore, constitutes a disadvantage of this method. But even more important than the cost of the method is the relative degree of its effectiveness. In addition to the relatively greater cost, there are two other serious objections to microscopic inspection which arise from defects inherent in the method. Specimens of pork containing trichinae may pass inspection undetected. This is particularly possible in meat containing young unencysted larvae or larvae enclosed within noncalcified cysts, since the larvae and the uncalcified cysts both are more or less transparent and therefore often difficult to recognize. The age of the vast majority of hogs which now come to slaughter in this country is less than a year, and at this age calcification of the trichina cysts is almost never present. Another valid objection lies in the large possibility that the particular specimen of

6. Schwartz, B.: Trichinosis in Swine and Its Relationship to Public Health, Ann. Rep. Smithsonian Inst. (1939), 1940, pp. 413-435.

meat examined may be free from trichinae, while the larvae are abundantly present in other portions of unexamined meat from the same animal. Thornbury [7] investigated hogs for the presence of trichinae in the diaphragm and muscles of the neck and loin. Among 1,043 hogs that were found trichinous the diaphragm was involved most often and most heavily, and yet no trichinae were found in the diaphragm in 24 per cent of these trichinous hogs. A fourth objection to the use of microscopic inspection is that the public may gain a false sense of security in assuming that pork which has passed inspection is safe for consumption in the raw state.

FEEDING OF COOKED GARBAGE TO HOGS

The second plan of control of trichinosis, that of cooking garbage which is to be fed to hogs, is one which has been adopted in Canada, England, the Hawaiian Islands and the states of Kentucky, Oregon and New York. This method has been repeatedly advocated by Hall [8] and by Wright.[8] The cost of instituting the method and providing apparatus for the cooking of garbage which is to be fed to hogs need not concern us. The points of fundamental importance are the effectiveness and the practicability of such a plan in the control of trichinosis. If all garbage was to be cooked prior to feeding it to hogs, the incidence of trichinosis among hogs so fed would be greatly decreased. However, it must be remembered that even among grain-fed hogs approximately 0.9 per cent of the animals are trichinous [9] and that the method under discussion would not eliminate the infection in grain-fed hogs. The method of feeding cooked garbage to hogs could undoubtedly be enforced in the case of large swine raisers, but it appears practically impossible to enforce the law in the case of small swine raisers. This fact alone makes the method, from a practical point of view, unworkable. In the states of New York, Oregon and Kentucky difficulties of enforcement have rendered the law ineffective. Furthermore, in the state of Oregon a person is permitted to feed swine garbage which has accumulated from his own household. In Chile, where the principal method of control is microscopic inspection, the law wisely forbids the feeding of any garbage, cooked or uncooked, to hogs.

The successful operation of this method of control would require a huge corps of sanitary inspectors and a tremendous job of policing. The method is regarded as less practical than microscopic inspection.

PROCESSING OF PORK

The third possible method of control is that of processing pork and pork products. By processing is meant the treatment by methods of freezing, cooking, smoking, curing or other means, according to federal specifications, which will render nonviable any trichinae present in meat. It is proposed that the federal government enact legislation which will require all pork that is intended for interstate shipment to be so processed as to render it free from living trichinae; furthermore, that all pork, pork products or meat preparations containing pork be required to bear the following label: "This product conforms to U. S. specifications for processing of pork." Likewise it is proposed that all city, county or state health authorities adopt similar regulations governing the preparation of all pork which is intended for local sale within the city, county or state, and that all such pork shall be labeled "This product conforms to (local) health regulations for processing of pork."

Trichinae are killed at a temperature of 55 C., but the federal regulations require that in killing trichinae by heat all parts of the meat shall be raised to a temperature of 58.33 C. (137 F.). In the case of fresh pork, processing consists principally in the methods of freezing. Trichinous meat in sections of 6 inches or less can be rendered noninfective by refrigeration [10] at a temperature of 5 F. for twenty days, minus 10 F. for ten days or minus 20 F. for six days. It has also been found [11] that raw pork in commercial quantities may be rendered free of infective trichinae by lowering its temperature to minus 35 C. (minus 31 F.) or by freezing [12] at minus 17.8 C. (0 F.) for seventy-two hours. In ground meat, encysted trichinae are killed within a few minutes at a temperature of minus 17.8 C. (0 F.). In most deep-freeze cabinets, such as those used in the home, temperatures varying from 0 to 5 F. are usually maintained. The main costs connected with this method of control of trichinosis are the costs of apparatus, such as refrigerating units, and of storage space. These expenses are initial ones, and similar initial expenses would be found necessary in any other method. The operation or maintenance of the method, however, would involve relatively little personnel as compared with microscopic inspection, and the method of processing therefore would be much cheaper. In the last analysis the cost of this method would not be great and would, of course, be borne by the consumer. The consumer would, in fact, be glad to assume this extra cost if he could have the assurance that he was receiving meat that was free from living trichinae.

The method of processing pork would largely eliminate trichinosis from hogs very quickly and would at the same time check the spread of trichinosis to man. If in any one year all pork should be processed, all trichinous pork would contain only nonviable trichinae, and any scraps of such pork which might reach the new crop of hogs would be incapable of producing trichinosis in them. Butcher hogs that come to market average from 9 to 10 months of age, sows average from 20 to 24 months, and the overall average age of hogs at the time of slaughter is about 11 months.[4] It is logical, therefore, to expect a rapid decrease in the incidence of trichinosis in hogs within a very few years after the general introduction of such a method of control. The hog raiser and the packer would benefit greatly from the advantages which would accrue to them in being enabled to supply to the public pork which was free from its chief stigma, the danger of trichinosis.

The initiative for such a method of control appears, from past indications, to rest primarily on physicians and public health authorities. The time will surely come when the public will insist that the pork which it eats, no less than the milk which it drinks, is safe for human consumption.

7. Thornbury, F. J.: The Pathology of Trichinosis: Original Observations, Univ. M. Mag. **10:** 64-79, 1897.

8. Wright, W. H.: The Epidemiology of Trichinella Spiralis Infestation and Measures Indicated for the Control of Trichinosis, Am. J. Pub. Health **29:** 119-127, 1939; A Survey of Municipal Garbage Disposal Methods as Related to the Spread of Trichinosis, Pub. Health Rep. **55:** 1069-1077, 1940.

9. Schwartz, B.: Trichinosis in Swine and Its Relationship to Public Health, J. Am. Vet. M. A. **92:** 317-337, 1938.

10. Ransom, R. H.: Effects of Refrigeration on the Larvae of Trichinella Spiralis, J. Agric. Research **5:** 819-854, 1916.

11. Augustine, D. L.: Effects of Low Temperatures on Encysted Trichinella Spiralis, Am. J. Hyg. **17:** 697-710, 1933.

12. Blair, J. B., and Lang, O. W.: Effect of Low Temperature Freezing on the Encysted Larvae of Trichinella Spiralis, J. Infect. Dis. **55:** 95-104, 1934.

Clinical Notes, Suggestions and New Instruments

ACCIDENTAL INTRA-ARTERIAL INJECTION OF OXOPHENARSINE HYDROCHLORIDE (MAPHARSEN)

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No reference to the effects of the intra-arterial injection of oxophenarsine hydrochloride (mapharsen) was found in the textbooks and literature available. Stokes[1] implied that arsphenamine may be safely injected intra-arterially but did not mention that there may be reactions from the other arsenicals. The painful reaction described here is believed to have followed such an accidental injection.

A brown Panamanian aged 22 was under treatment for early latent syphilis at the Venereal Disease Clinic of Gorgas Hospital, Ancon, C. Z. Both the Wassermann and Kahn reactions of the blood serum were strongly positive, but there was no other clinical or laboratory evidence of disease. The reaction followed his twelfth injection of oxophenarsine hydrochloride.

On the regular weekly clinic visit, a routine check-up serologic test and 0.05 Gm. of oxophenarsine hydrochloride were ordered. The nurse injector, using a 22 gage needle and a 10 cc. syringe, withdrew a 4 cc. blood specimen from a vessel in the left antecubital fossa. Releasing the tourniquet, but leaving the needle in place, she detached the syringe containing the blood sample, attached a second syringe containing the 0.05 Gm. of oxophenarsine hydrochloride in 8.6 cc. of distilled water and injected the drug in approximately ten seconds.

Before completion of the injection, the patient complained of pain in the hand and lower part of the forearm. Within a few seconds after the withdrawal of the needle the pain had increased to such an extent that he was groaning, his face became pale and he seemed about to faint. It was necessary to assist him to a bed in the clinic. Morphine sulfate 0.015 Gm. was given for the pain about five minutes after the injection. One half hour later the patient was still moaning with pain, and he was transferred to a hospital ward, where another 0.015 Gm. of morphine was given.

When seen by one of us (J. L. W.) about one minute after the injection, the skin of the forearm and hand was bluish. This dusky color faded in a few minutes and the skin of the hand became slightly pinker than normal. The pain was described as a steady boring or aching pain and not as burning or cramplike. It was generalized in the forearm and hand but was most severe in the thenar and hypothenar eminences and in the finger tips. These areas were slightly tender to touch. Motion and tactile sensation were unimpaired. Local applications of heat or cold did not relieve the pain.

Two hours later the pain was considerably less. The skin color and pulse were normal, but the forearm and hand were slightly swollen as compared to the opposite extremity. Five hours after the injection the oral temperature was 99.2 F. and slight swelling was still present but there were no other changes. The following day the patient had no complaints and was discharged from the hospital. He has been followed in the clinic for one year without developing sequelae and has received subsequent injections of oxophenarsine hydrochloride without difficulty.

We believe that the reaction was due to an intra-arterial injection of oxophenarsine hydrochloride for the following reasons:

1. The color of the blood specimen drawn from the patient was a much brighter red (arterial) than the specimens drawn from other patients at about the same time. This difference was still noticeable when we examined the rack of blood specimens one and one half hours later.

2. The pain began immediately and occurred only distal to the site of the injection. There was no pain in the upper arm as with venous spasm. There was no swelling or pain at the injection site suggesting an infiltration.

3. The pain was generalized in the forearm and hand and was not confined to an area corresponding to the distribution of a nerve.

4. On examination of the patient's antecubital fossa, a superficial pulsating vessel could be felt just beneath and parallel to the visible vein that the operator had intended to use.

The injector was relatively inexperienced, so that minor abnormalities in the injection procedure such as the resistance of the vessel wall, the ease with which the blood was withdrawn and the color of the blood may have been overlooked. The small size of the needle (22 gage) also tends to minimize the first two points. One possible explanation for the fact that blood did not spurt from the needle during the interval when the syringes were changed is that the bevel of the needle was in contact with the vessel wall.

In the two years that the clinic has been in operation, 60,000 intravenous arsenical injections have been given. During this time there has been one other similar but less severe reaction. Subsequent examination showed a superficial artery in the forearm of this patient also, but there was no other evidence to confirm the suspicion that the pain was due to an intra-arterial injection.

SUMMARY

A severely painful local reaction followed an injection of oxophenarsine hydrochloride. The pain lasted several hours and required morphine for relief. We believe it was due to an accidental intra-arterial injection because a blood specimen drawn just before the injection was bright red (arterial) blood, the pain was immediate and entirely distal to the point of injection and an anomalous superficial artery was found at the injection site. Except for the severe pain, there were no serious sequelae.

From the Branch Medical Clinic, Gorgas Hospital, Ancon, Canal Zone.

1. Stokes, John H.; Beerman, Herman, and Ingraham, Norman R., Jr.: Modern Clinical Syphilology, ed. 3, Philadelphia, W. B. Saunders Company, 1944, p. 401.

Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

AUSTIN SMITH, M.D., *Secretary.*

PENICILLIN (See New and Nonofficial Remedies, 1945, p. 214).

The following additional dosage forms have been accepted:

MERCK & CO., INC., RAHWAY, N. J.

Penicillin Sodium: Vials containing 200,000 Oxford Units.

Penicillin Calcium: Vials containing 200,000 Oxford Units.

SULFADIAZINE (See New and Nonofficial Remedies, 1945, p. 185).

The following dosage form has been accepted:

VOGEL LABORATORIES, MOHEGAN LAKE, N. Y.

Emulsion Sulfadiazine 5% Sterilized: 50 cc. and 200 cc. bottles. A 5 per cent suspension of sulfadiazine in an emulsion of beeswax, liquid petrolatum, triethanolamine and water.

DIPHENYLHYDANTOIN SODIUM (See New and Nonofficial Remedies, 1945, p. 528).

The following dosage form has been accepted:

PREMO PHARMACEUTICAL LABORATORIES, INC., NEW YORK

Capsules Diphenylhydantoin Sodium: 0.03 Gm. and 0.1 Gm.

THEOPHYLLINE ETHYLENEDIAMINE (See New and Nonofficial Remedies, 1945, p. 388).

The following additional dosage form has been accepted:

ERNST BISCHOFF COMPANY, INC., IVORYTON, CONN.

Solution Aminophylline: Ampuls 0.24 Gm. in 10 cc. and 0.48 Gm. in 2 cc.

Council on Physical Medicine

The Council on Physical Medicine has authorized publication of the following report. HOWARD A. CARTER, Secretary.

SAFETY OF MODERN ALTERNATING POSITIVE AND NEGATIVE PRESSURE RESUSCITATORS

HENRY SCHWERMA, PH.D.
AND
A. C. IVY, PH.D., M.D.

This investigation was undertaken primarily to ascertain whether one of the currently used positive and negative pressure resuscitators (so-called "suck and blow" type) when used for one to three hours will cause a significant amount of damage to the lung. The experimental conditions were such that we had the opportunity of observing whether the use of the resuscitator interfered with the return of a low blood pressure to normal as compared to intermittent positive pressure devices.

The subject requires investigation because resuscitators of the "suck and blow" type are being quite widely used in the United States [1] and because the wisdom of their use, particularly by laymen, has been questioned.[2] It is appropriate to examine the literature to ascertain the evidence on which the questionable use of the "suck and blow" resuscitator in based.

ANALYSIS OF THE LITERATURE

Henderson and Turner [3] state that "the deaths resulting from suck and blow respiration applied to dogs in the experiments of Henderson and Haggard,[4] of Henderson [5] and of Coryllos [6] deserve serious consideration." The reference to Henderson and Haggard [4] refers to four experiments in which it was observed that excessive artificial respiration for a prolonged period decreases blood pressure, washes out carbon dioxide and causes apnea vera. The reference to Henderson [5] refers to a number of experiments in which excessive artificial respiration for twenty-five to thirty minutes is followed by apnea so prolonged that the heart fails after seven or eight minutes for the lack of oxygen. However, in producing the hyperventilation the respiratory rate was 60 to 120 cycles per minute (the minute volume is not provided) with an alternating positive and negative pressure of 30 cm. of water. The reference to Coryllos [6] refers to experiments on 7 dogs which were under amytal anesthesia and received artificial respiration for one to eight hours. Four of the 7 dogs died from five to twelve hours after the conclusion of the experiment and "at autopsy relatively slight pulmonary and other lesions were found, which could not explain death." "Suspecting chilling as a possible cause," the other dogs were kept warm, the gas inhaled was humidified and no deaths occurred. Henderson [7] in 1916 was inclined to doubt "whether an active withdrawal of air with a pump of limited stroke has any very serious objections" and referred to his hyperventilation experiments [8] in which he used "two pumps arranged in a manner similar to the 'lungmotor' and worked quite violently" and which caused "no particular ill effects on the lungs." In the same paper he recommended that the "Pulmotor Model B" be equipped with blow off valves limiting the positive pressure to 15 cm. of water pressure and the negative pressure to 10 cm. Meltzer,[9] who studied the old "Pulmotor," found that it would frequently turn off the supply of air when the pressure in the trachea was only 4 cm. of water. As a result the lungs were inadequately ventilated and decreased in volume with the chest open. He believed that the suction would favor collapse of the bronchioles and hence only the dead air space would be ventilated. He pointed out that Henderson's favorable view of "suck and blow" was due to the fact that the pumps he used were directly connected to the trachea, which would obviate the resistance in the nasopharynx. Meltzer [9] used a device with bellows for intermittent positive pharyngeal insufflation with a tube in the stomach to release any gas entering it. In 1934 Henderson,[10] in discussing intratracheal insufflation with intermittent positive pressure, recommended a maximum pressure of 40 mm. of mercury for the adult and 25 mm. for the infant. In discussing the E and J Resuscitator he stated that "pressure and suction require intubation of the trachea, an operation that properly and legally can be performed only by physicians. The administration of pressure and suction with a mask sometimes works; but it is liable to fail when most needed. In the hands of laymen, such apparatus cannot be made free from serious danger of injury to the lungs." Henderson and Turner [3] in 1941 experimented with the E and J Resuscitator and gassed 6 dogs with carbon monoxide to the stage of coma, in which stage they were either convulsive or limp. The three treated with the E and J Resuscitator and 5 per cent carbogen (presumably 5 per cent carbon dioxide and 95 per cent oxygen; they do not state) died and the 3 treated with manual artificial respiration and carbogen survived. The lungs of the animals treated in these and other experiments were not examined. They found that, "in brief, a resuscitator is merely an inhalator which tends to impede natural breathing by sucking and blowing." They wrote however that, "in order to administer carbon dioxide efficiently, the pulmotor, E and J Resuscitator and similar devices should be modified to eliminate the suction phase, decrease the pressure of the blowing phase and greatly increase its volume. They would then become efficient resuscitation apparatus, which at present they are not." But we have been unable to find any reason given or evidence submitted by Henderson indicating why suction is undesirable.

We have been unable to find evidence in the publications of Henderson and his associates which indicates that the lung is damaged by alternating positive and negative pressure. The alternating positive and negative pressure of 30 cm. of water (22 mm. of mercury) used in his hyperventilation experiments may have been conducive to the fall in blood

From the Department of Physiology, Northwestern University Medical School, Chicago.

1. Ross, B. D.: A Survey of Methods for Artificial Respiration, J. A. M. A. **122**: 660 (July 3) 1943.
2. Drinker, C. K.: Restoration of Breathing in Emergencies and Maintenance of Respiration in Nonbreathing Patients, J. Oklahoma M. A. **37**: 285 (July) 1944. Henderson and Turner.[3]
3. Henderson, Yandell, and Turner, J. M.: Artificial Respiration and Inhalation, J. A. M. A. **116**: 1508 (April 5) 1941.
4. Henderson, Yandell, and Haggard, H. W.: J. Biol. Chem. **33**: 355, 1918.
5. Henderson, Yandell: Am. J. Physiol. **25**: 310, 1910.
6. Coryllos, P. N.: Surg., Gynec. & Obst. **66**: 698, 1938.
7. Henderson, Yandell: Resuscitation Apparatus, J. A. M. A. **67**: 1 (July 1) 1916.
8. Henderson and Haggard.[4] Henderson.[5]
9. Meltzer, S. J.: M. Rec. **92**: 1, 1917.
10. Henderson, Yandell: Resuscitation, J. A. M. A. **103**: 750 (Sept. 8), 834 (Sept. 15), 1934.

pressure and fatal apnea that he observed [11] or the fall in blood pressure may have been due to anesthesia or to both. Seevers and his associates [12] observed that hyperventilation (p_H increase in plasma 0.31 unit) decreased the systolic pressure 10 mm. of mercury before and 25 mm. of mercury during anesthesia. Apnea occurred for five to nine minutes but was followed by a return of normal respiration and blood pressure. This evidence indicates that acapnia, though undesirable, is not as serious as the experiments of Henderson and Haggard indicate. However, it does constitute one of the arguments in favor of the use of carbogen in the treatment of carbon monoxide anoxemia, in which there is no pronounced accumulation of carbon dioxide in the body as occurs in obstructive asphyxia. As Henderson and Turner have shown, artificial respiration of the suck and blow type interferes with the rhythm of natural breathing. But any type of artificial respiration which is not synchronized with natural breathing will do the same. The evidence of Henderson and Turner regarding the comparison of the effectiveness of manual with mechanical artificial respiration, in which only 6 gassed dogs were used, is certainly inadequate. The results of such a comparison made by us on larger groups of dogs will be submitted in our subsequent report.

THE PRESSURE REQUIRED TO RUPTURE ALVEOLI

The amount of pressure required to rupture alveoli has been studied by several groups of investigators. Ewald and Kobert,[13] using rabbits, found that air in the lungs under a pressure of 40 mm. of mercury (54.4 cm. of water) caused rupture of the alveoli and pneumoperitoneum. In 1931 Joannides and Tsoulos,[14] using dogs, found that a continuous intrapulmonic pressure of 60 mm. would cause interstitial emphysema and that higher pressure would cause pneumothorax, pneumoperitoneum and air embolism. Polak and Adams [15] placed a gas trap in the carotid artery of dogs and found that air appeared in the trap when the intrapulmonic pressure was 80 mm. of mercury (109 cm. of water) for ten second periods of inflation; it did not appear when the pressure was 60 mm. of mercury; 100 mm. of mercury caused extensive interstitial mediastinal and subcutaneous emphysema. Burkhardt and Ivy [16] found that the presence of 100 mm. of mercury pressure of air in the trachea for two seconds will cause rupture of the pleura in some dogs as well as emphysema. Macklin [17] has described anatomically the passage of air bubbles from the alveoli along the sheaths of the pulmonary vessels to the mediastinum when the lungs are overinflated. Coryllos,[6] studying the isolated lung of dogs, observed the leakage of air when the continuous intrapulmonic pressure was 52 mm. of mercury.

More recently studies have been performed the results of which reveal the occurrence of interstitial and mediastinal emphysema with lower intrapulmonic pressures. These investigators were interested in the possible dangers of intratracheal anesthesia under the conditions of thoracic surgery. Marcotte and his associates,[18] using 11 dogs, introduced air as a continuous stream into the trachea through a catheter with an outflow. The intrabronchial pressure was maintained at pressures ranging from 16 to 110 mm. of mercury for eighteen to seventy-five minutes. A sustained pressure of 24 mm. of mercury (32.6 cm. of water) produced interstitial and mediastinal emphysema, but such a lesion was not obtained with 18 mm. of mercury until the chest was opened. Cats were more susceptible, developing emphysema with pressures of 16 to 20 mm. of mercury. Rasmussen and Adams [19] conducted acute and chronic experiments on dogs to ascertain the effects of an intermittent type of positive pressure on the lungs. The on-period of the pressure in each cycle was three times longer than the off-period in order to simulate the longer expiratory pressure period present during an attack of asthma. The lungs were inflated twenty-eight times per minute with a pressure at the carina ranging from 30 to 35 mm. of mercury. The acute experiments varied from thirty-five to seventy-five minutes in duration. In the chronic experiments the lungs were inflated, with the animal under morphine anesthesia, for fifteen minutes twice a week for from one week to eleven months. Microscopically all the dogs had slight to pronounced interstitial emphysema. Two, or 22 per cent of the 9 dogs, died with mediastinal emphysema, air embolism and subpleural hemorrhage during the period of overinflation. During overinflation the arterial pressure was much reduced in the acute and cyanosis occurred in the chronic experiments.

It should be emphasized that in the foregoing experiments the pressures were intrabronchial and not oronasal and that they were relatively high and more or less continuous. Orth and his associates [20] found that the fall of pressure from a mask to the carina was from +34 mm. of mercury at the mask to +28 mm. of mercury at the carina when he used a resuscitator which gave the largest positive pressure at the mask. It would appear from the observations of Rasmussen and Adams and of Marcotte and his associates on the dog that the positive pressure at the mask should probably not exceed 20 mm. of mercury (27 cm. of water). If the device has a blow off valve set at a maximum of 20 mm. of mercury, any lack of synchronization with natural breathing when it appears unexpectedly will safeguard the lungs from undue pressure. It is assumed that, when natural breathing is adequate, artificial respiration will be stopped and the inhalator turned on.

INTRABRONCHIAL PRESSURE IN MAN UNDER ORDINARY CONDITIONS

Under ordinary breathing the intrabronchial pressure varies under +1 to +5 mm. of mercury to —1 to —3 mm. of mercury. During speaking with the ordinary pitch and loudness, the intratracheal pressure ranges from 10 mm. of mercury to 18 mm. of mercury. During shouting it may be as large as 70 mm. of mercury. For a second or more during coughing, the intratracheal pressure may be as high as 80 to 100 mm. of mercury.[21] In the dog, during expiration with the

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trachea obstructed, the pressure may be as high as +76 mm. of mercury and during inspiration as low as —35 mm. of mercury. This must be taken into account in interpreting the presence of interstitial and mediastinal emphysema and air embolism in obstructive asphyxia.

LACK OF EVIDENCE OF LUNG INJURY USING THE POSITIVE-NEGATIVE PRESSURE RESUSCITATOR

Relatively little work has been published in which the effect on the lungs of a resuscitator of the suck and blow type has been investigated.

Rost [22] in 1932 used a suck and blow type of artificial respiration in which the peak positive and negative pressure was ±14 mm. of mercury. The animal was anesthetized and curarized. No deleterious effects on the lung or blood pressure were observed in acute or recovery experiments. He observed no untoward effects when the technic was applied to the human being. The lungs were not examined for emphysema microscopically. Coryllos [6] used the E and J Resuscitator, which provides a maximum positive pressure of 14 mm. of mercury and a negative of 9 mm. of mercury. He obtained favorable results with its use in human and canine tests. Of the 4 dogs which died, he reports only one microscopic study of the lungs and does not mention emphysema. In his other tests, if emphysema occurred it was not significant. Birnbaum and Thompson [23] in a series of studies summarized by Thompson [24] in 1944 used the Emerson Resuscitator, which is a suck and blow type (+14 mm. of mercury and —9 mm. of mercury). They chiefly studied resuscitation from obstructive asphyxia and anoxia due to the inhalation of inert gases, such as pure nitrogen. They resuscitated 72 of 106 animals. They did not study the lungs microscopically, but it was probably not necessary because they used the resuscitator only a few minutes. Steinberg and Dietz [25] used the McKesson resuscitator, which inflates and deflates the lungs by a fixed volume, on seven dogs for from one to eight hours. They do not provide the peak positive and negative pressures used. The microscopic study of the lungs in 2 of the animals showed no abnormalities except some hyperemia. More recently Drinker [2] has stated "I have never been afraid that blow and suck machines as safeguarded today would do harm by rupturing the lungs. Their failure is through lack of air delivery." In 1923 he and his associates [26] reported experiments on cats in which a lungmotor set at +20 mm. of mercury and —4 mm. of mercury did not embarrass the dilated heart in carbon monoxide poisoning and produced no evidence of rupture of the lung. They say that "the possibility that positive ventilation might rupture a diseased lung can never be dispelled by animal experimentation." In his latest paper Drinker [2] reports an experiment on a dog under anesthesia and curare in which a suck and blow apparatus was connected to a tracheal cannula. The positive pressure was 10 cm. of water and the negative 8 cm.; air was used. As a consequence he observed during a period of four hours a gradual decrease in lung volume. It is stated that "the loss is due to gradual atelectasis and exudation in dependent parts of the lungs where expansion is most difficult. It occurs more rapidly in a machine which sucks during expiration than in one in which expiration is passive, as in normal breathing." This would seem to be reasonable under the condition of the experiment, but more evidence is needed to establish the latter statement. It may not apply when 5 or 7 per cent carbogen is used but may when oxygen is used; or it may apply only if the minute volume of respiration is kept constant by a tight fitting tracheal cannula. He interprets his observations as arguing against the prolonged use of the suck and blow method, especially in the presence of water or exudate in the air passages.

Thus, our review of the literature has revealed no evidence showing that lung damage occurs in the dog when continuous insufflation yielding an intrabronchial pressure of 18 mm. of mercury or less is used or when the modern suck and blow resuscitators with a peak of positive and negative pressure of +14 mm. of mercury and —9 mm. of mercury is used. The evidence, however, is meager.

DO RESUSCITATORS PREDISPOSE TO PNEUMONIA?

Another point in connection with lung damage deserves consideration: One would like to know whether the methods used for resuscitation vary in regard to the predisposition to subsequent pulmonary disease. If one method predisposes more to atelectasis and pulmonary edema than another, this might predispose to pneumonia. Or, if a method causes the passage of more material from the upper respiratory tract into the lungs, this might predispose to pneumonia. The first possibility is suggested by the aforementioned experiment of Drinker.[2] The second possibility was studied by Drinker and his associates [26] in 1923. In these experiments they compared the extent to which material dripped into the trachea of anesthetized animals is carried into the lungs under natural breathing and artifical respiration. They concluded from the experiments that "apparently, if one utilizes an air blast which is sufficiently vigorous to get air into the alveoli in anything like normal amounts, he is in danger of carrying in material from the upper respiratory tract." Experiments with manual artificial respiration were not conducted. It would seem that the only way to obtain evidence on this problem would be to asphyxiate animals, resuscitate some by manual respiration and others by a positive pressure method, and ascertain the incidence of pneumonia in the two groups.

IS A NEGATIVE PHASE IN THE RESPIRATORY CYCLE OF RESUSCITATORS ADVANTAGEOUS?

Though we have been unable to find evidence showing that intermittent positive-negative intrapulmonic resuscitators damage the lungs or interfere with the circulation, the question arises What are the advantages of adding the negative phase to the respiratory cycle, if any? Birnbaum and Thompson [27] presented evidence indicating that suction is beneficial for the reason that it stimulates inspiratory sensory fibers in the vagi. This is the only suggestion that has been made indicating that suction directly affects respiration other than that suction assists respiration in the presence of hypotonic muscles associated with feeble respiration or the absence of respiration. Comroe and Dripps,[28]

22. Rost, E.: Ztschr. f. d. ges. exper. Med. **82**: 255, 1932.
23. Thompson, S. A., and Birnbaum, G. L.: Surg., Gynec. & Obst. **74**: 1078, 1942. Thompson, S. A., and Birnbaum, G. L.: Surgery **12**: 284, 1942. Birnbaum, G. L., and Thompson, S. A.: J. Thoracic Surg. **12**: 607, 1943. Thompson, S. A., and Birnbaum, G. L., ibid. **12**: 624, 1943. Birnbaum and Thompson.[27]
24. Thompson, S. A.: Ann. Surg. **120**: 94, 1944.
25. Steinberg, B., and Dietz, A.: J. Lab. & Clin. Med. **29**: 695, 1944.
26. Drinker, C. K.; Drinker, K. R.; Shaw, L. A., and Redfield, A. C.: J. Indust. Hyg. **5**: 109, 1923.
27. Birnbaum, G. L., and Thompson, S. A.: Surg., Gynec. & Obst. **75**: 79, 1942.
28. Comroe, J. H., and Dripps, R. D., Jr.: Ann. Rev. Physiol. **7**: 653, 1945.

however, suggest that "a moderate weight applied to the chest wall will probably serve as well without incurring the risks involved with suction." It might be rejoined that the "moderate weight" would increase the amount of positive pressure required and, it might be asked, What risks does —8 mm. of mercury of suction introduce when applied at a mask with an "airway" in position? Nevertheless, in our opinion, the observations of Thompson and Birnbaum call for confirmation.

Maloney [29] has suggested that expiration by suction removes noxious gases better than passive expiration. This would be difficult to prove unless the minute volume of respiration is carefully controlled.

Birnbaum and Thompson [27] suggest that resuscitators of the suck and blow type by "milking" blood from the lungs significantly improve the return of blood to the heart. This suggestion could not be substantiated by Volpitto, Woodbury and Abreu.[30] These investigators found that, "irrespective of the method of mechanical artificial respiration employed, recovery of the animal was accomplished when respiratory arrest and slow weak cardiac contractions were produced with helium" inhalation. They also found that the effect of the various mechanical methods on the pulmonic and systemic blood pressure was essentially the same.

While our work was in progress a paper by Orth and his colleagues [20] appeared in which they reported results on the effect of artificial respiration on the lungs. Using fewer animals than we have employed, they arrived at essentially the same conclusion, which is warranted by the results of our investigation. Their conclusion was as follows: "Pulmonary damage was present in control groups of animals either sacrificed and examined without any period of anesthesia or respiratory manipulations or subjected only to anesthesia in which respiration occurred spontaneously. Similar damage was evident in groups of animals subjected to various methods of artificial respiration."

THE EXPERIMENTAL DESIGN

Four series of experiments were performed. Series A consisted of 102 dogs which were used as a control series to ascertain the incidence of pulmonary disease in laboratory dogs as they were selected by us for this study during the late fall and winter months. Series B consisted of 25 dogs which were used to study pulmonary disease after use of the E and J Resuscitator for from one to three hours when the respiratory muscles were paralyzed with curare and the animals under anesthesia. Series C consisted of 5 dogs which were used to ascertain whether the use of the resuscitator in the presence of natural breathing would cause emphysema. Series D consisted of 22 dogs which were used to ascertain whether the use of other resuscitators damaged the lungs or interfered with blood pressure.

The dogs weighed 5 to 12 Kg. All were "clinically normal" as judged by appearance, behavior and the absence of the signs of distemper or other disease. X-ray films of the chest were not made because of the scarcity of x-ray film. Physical examination of the chest in the untrained dog is not satisfactory.

A. *The Control Series.*—One hundred and two dogs served as a control series; they were killed either by bleeding during pentobarbital anesthesia or at the end of an experiment of two hours' duration under anesthesia with natural breathing. The lungs and trachea of these dogs were then examined grossly and all lesions were measured, described and recorded.

B. *The Treated for Recovery Series.*—Twenty-five dogs served as the "treated series." They were anesthetized with pentobarbital and the femoral artery was connected to an apparatus for recording blood pressure. Respiratory movements were recorded by means of a pneumograph held in place by a towel at the level of the xiphoid. The femoral vein was cannulated to facilitate the injection of curare along with 0.9 per cent solution of sodium chloride. In several of the earlier experiments a tape measure was fixed in place about the lower part of the chest to measure the excursion during natural breathing and later artificial respiration. In several of the earlier experiments the parietal pleura at the fifth interspace was exposed to observe the movement of the lungs during natural and artificial respiration through the parietal pleura without opening the thorax. Between observations this wound was snugly covered with gauze held in place beneath the skin with clips.

The artificial respiration was given through a mask of the type commonly employed for the study of oxygen consumption in unanesthetized dogs. The fit of the rubber diaphragm of the mask about the neck was such that leaks were absent or insignificant and did not interfere with the effectiveness of the artificial respiration. The positive and negative pressure in the mask was observed by a sensitive aneroid pressure gage recording in ¼ inch of water pressure.

The E and J. Resuscitator was used for artificial respiration. It is designed to provide a gas for inhalation (an inhalator) or a gas for positive and negative pressure artificial respiration. The maximum positive pressure provided is 14 mm. of mercury (19 cm. of water); the maximum negative pressure is 9 mm. of mercury (12 cm. of water). These pressures, of course, obtained in all of the twenty-five experiments. The apparatus was set to cycle fourteen or fifteen times per minute. The volume of gas flow per minute, as measured by a Precision flow meter introduced in the circuit in dogs of various weights, varied from 300 to 500 cc. per minute. In a dog weighing 9 Kg., for example, the minute volume of ventilation, using 100 per cent oxygen, was 400 cc. In the first 10 dogs 100 per cent oxygen was used and in the last 10 dogs 3 per cent carbogen (3 per cent carbon dioxide and 97 per cent oxygen), and in 5 both 100 per cent oxygen and 7 per cent carbogen.

The E and J Resuscitator was chosen because it has both positive and negative pressure. It was assumed that if the apparatus caused no lung damage or deleterious circulatory effects the results would apply not only to other "suck and blow" devices with the same maximum pressures but also to intermittent positive pressure devices having the same maximum positive pressure.

After making control observations for fifteen or twenty minutes, curare was injected to paralyze the muscles of respiration. A dose of 0.5 cc. per kilogram of body weight of Merck's curare solution number 569 was adequate usually to paralyze completely or almost completely the diaphragm and intercostal muscles. Occasionally additional amounts were required. In an occasional dog in which very feeble movements persisted, doses of 2 cc. per kilogram of body weight were

29. Maloney, A. H.: Anesth. & Analg. **23**: 39, 1944.
30. Volpitto, P. P.; Woodbury, R. A., and Abreu, B. E.: Influence of Different Forms of Mechanical Artificial Respiration, J. A. M. A. **126**: 1066 (Dec. 23) 1944.

ineffective. Larger doses were not given because we did not consider complete paralysis necessary for our purpose. The resuscitator was started after the curare was injected and at the point of almost complete paralysis of the muscles of respiration. At fifteen minute intervals the resuscitator was turned off and the inhalator on for one or two minutes to ascertain the state of activity of the respiratory muscles. This was done because Stewart [31] has observed that at times during resuscitation of the respiratory center from anemic anoxia with the vagi intact the rhythm of the artificial respiration is impressed on the respiratory center.

Appreciable endogenous respiratory movements occurred after from thirty to ninety minutes. In half of the dogs it occurred between thirty and sixty minutes and in the remainder after sixty minutes (table 2). When endogenous movements returned, the artificial respiration was continued except for brief periods until the animal could maintain the oxygenation of the blood sufficiently to allow recovery. This means that all the dogs received artificial respiration for from one up to three hours. We did not maintain the animals longer because we believed that "suck and blow" devices will be ordinarily used for resuscitation for a period of less than an hour.

When the animal's natural breathing was adequate, the wounds were aseptically closed, 10 mg. of ephedrine was injected subcutaneously and the animal was kept warm until it recovered from the anesthesia. (The animal was also kept warm during the experiment.) The animals were killed three days later by the following procedure: The animal was anesthetized with pentobarbital. The trachea and carotid artery were exposed. The animal was then bled and the trachea clamped at the end of inspiration. The chest was then carefully opened to ascertain whether pneumothorax or collapse of a lung was present. The lung was then examined, including a search for evidence of interstitial and mediastinal emphysema; sections were taken for microscopic examination in a few instances.

Only 1 dog was killed immediately after the period of the artificial respiration; he had been under the artificial respiration for over two hours. There was no evidence of interstitial or mediastinal emphysema. Other animals in this group were not studied because we were interested only in whether damage of a significant degree would occur, because it did not occur in the experiments of Marcotte and his colleagues,[18] who used 18 mm. of mercury of continuous intrabronchial pressure for eighteen to seventy-five minutes in the dog, and because we desired to look for emphysema under a somewhat more rigorous condition.

C. In order to ascertain whether interstitial or mediastinal emphysema might occur under the pressure conditions imposed by the E and J Resuscitator in the presence of natural breathing, the following method was used:

The dogs were anesthetized with pentobarbital and fitted with a pneumograph. The mask was fitted and a control record of respiration was made. Then the E and J Resuscitator was started with 100 per cent oxygen and kept operating for one hour; after that, 7 per cent carbogen was administered for ten minutes. The dog was then killed by bleeding and the trachea clamped at the end of an inspiration. The chest was carefully opened, as stated before; the lungs and mediastinum were inspected with a 16 diameter magnifying lens, and sections were taken immediately for microscopic study.

D. Experiments were also performed in which the return of blood pressure to normal after the injection of curare was studied in relation to the type of resuscitator used. We used (*a*) the E and J Resuscitator, (*b*) an intermittent positive pressure resuscitator with some continuous insufflation at 10 mm. of mercury pressure, (*c*) the G. E. Pneumolator, which is a positive pressure (14 mm. of mercury) resuscitator, and (*d*) the Drinker respirator for infants.

RESULTS

The pathologic findings are summarized in table 1.

A. *Control Series.*—Of the 102 dogs in the control series 15, or 15 per cent, had a patch or patches of consolidation varying from 1 cm. in diameter up to as many patches as would yield an estimated total of 33 per cent consolidation. The consolidation ranged from 10 per cent to 30 per cent in 7 of the 15 dogs, or 7 per cent of the 102 dogs. Sixteen, or 15 per cent, of the remaining had minor pathologic changes consisting of petechial or small ecchymotic hemorrhages, scars or marginal emphysematous blebs. Seventy-one had perfectly normal lungs.

This shows that in the absence of a careful physical examination of the chest, or a roentgenogram, during the winter months 7 per cent of dogs can have an appreciable bronchopneumonia and appear "clinically normal."

B. *Treated for Recovery Series.*—Two dogs died, 1 at forty-eight and the other at seventy-eight hours after recovery from the experiment; both had bronchopneumonia. The one that died at forty-eight hours had an area of consolidation measuring 12 by 8 by 4 cm. The central portion of the area manifested gray hepatization. The lobe had other small patches of bronchopneumonia. The one that died at seventy-two hours had an area of consolidation of 6 by 4 by 4 cm. and other small patches of bronchopneumonia. This dog had some empyema and pneumothorax, which was due to the rupture of three marginal emphysematous blebs at the margin of the consolidated area described. There were areas of gray hepatization in the consolidated area. The presence of gray hepatization strongly indicates that pneumonia had been present for five days or was present at the time of the experiment. This interpretation is sustained by the findings on the control series. Minor changes were found in 4 (16 per cent) of the dogs.

Considering the control series, pathologic findings in this group cannot be attributed to the treatment. The study of the sections simply confirmed the gross diagnosis. The treatment may have aggravated the existing pneumonia, but this is not proved, and a very large series would be required to prove it. Joannides [32] reports that pneumonic, consolidated, dark colored lungs when subjected to increased intrapulmonic air pressure (25 to 35 mm. of mercury in the dog) either inside or outside of the body became crepitating and pinkish red again. He does not recommend that positive pressure be used in pneumonia, however.

C. *Treated During Natural Breathing.*—Interstitial or mediastinal emphysema was not found by microscopic examination or by examination under a magnifying lens in the dogs given artificial respiration during natural breathing.

31. Stewart, G. W.: Am. J. Physiol. **20**: 407, 1907.

32. Joannides, Minas: Insufflation of Compressed Air in the Treatment of Pneumonia, Arch. Int. Med. **47**: 196 (Feb.) 1931.

In this group of experiments it was found, as in the foregoing experiments, that some dogs during natural breathing followed the artificial respiration and some did not. At times the same dog would follow the rhythm of the machine and then again would not, as shown in the tracing. In no instance was the lack of synchronization between natural breathing and the machine serious.

TABLE 1.—*Incidence of Lung Abnormalities in Resuscitated Dogs Compared with Dogs Which Received No Resuscitation Treatment*

Lung Condition	Resuscitated Dogs		Control Dogs	
	Number	%	Number	%
Bronchopneumonia	2	8	15	15
Minor pathologic changes	4	16	16	15
Normal	19	76	71	70
Total	25	100	102	100

The only evidence of interference was a notch in the normal respiratory curve. The minute volume of ventilation remained the same except when the natural respiratory excursion was small; then the minute volume was greater with the machine.

D. *Observations on the Circulation with Three Resuscitators and One Respirator.*—The curare was injected rather rapidly with the idea of obtaining a fall in blood pressure so that the effect of the various methods of artificial respiration on the return of blood pressure could be ascertained. After the injection of curare the blood pressure fell to a level which ranged from 30 to 60 mm. of mercury. It remained at this level for a variable period, usually four to ten minutes, and then slowly rose. As a general rule the blood pressure had returned approximately to the control level at the time spontaneous respiration returned. But, as stated before, the artificial respiration was continued until the spontaneous respiration was adequate to maintain the animal, since we desired to have the animal recover for postrecovery observation.

The averaged results on the return of blood pressure are shown in table 2. The results show that the time

TABLE 2.—*A Comparison of the Rate of the Return of a Low Blood Pressure Due to Curare When Various Devices for Artificial Respiration Are Used*

Type of Resuscitator Used	Number of Experiments	Blood Pressure		Return of Spontaneous Respiration Time, Min.	Pressure Respiratory Cycle	
		Before Curare, Mm. Hg	After Curare, Mm. Hg		Positive, Mm. Hg	Negative, Mm. Hg
E & J Resuscitator	20	126	100	42	14	8
G. E. Positive	7*	130	104	60	14	0
B. Positive	8	136	100	33	10	0
Drinker Respirator	7	128	108	41	8	0

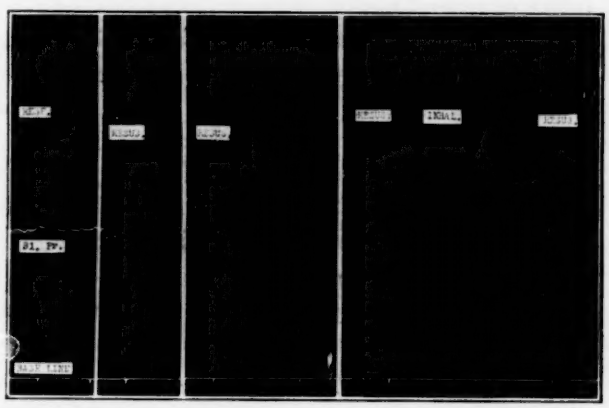
* One dog was killed when, at about two hours, it appeared that the blood pressure would not return to the control level; that may have been due to an overdose of pentobarbital.

for the return of blood pressure to normal did not vary significantly when the E and J Resuscitator, the G. E. Pneumolator (intermittent positive pressure), the B. Resuscitator (intermittent positive pressure with some continuous insufflation) or the Drinker Respirator (infant type) was used. We interpret the results as showing that neither of the types of artificial respiration with the pressures employed interfered significantly with venous return. If a difference does exist, many animals would have to be used to prove it because of the variation which exists in different animals.

SUMMARY AND CONCLUSIONS

1. One hundred and two "clinically normal" dogs were studied for the incidence of pulmonary disease. The chest was not examined carefully for physical findings because this is difficult in the untrained animal. X-ray films of the chest were not made because of the scarcity of film. Fifteen per cent of the animals had bronchopneumonia, the areas of consolidation ranging from 10 to 30 per cent in 7 per cent of the dogs. Fifteen per cent of the animals had minor pathologic changes, such as petechial or small ecchymotic hemorrhages, scars or marginal emphysematous blebs. In the remainder (70 per cent) the lungs were normal.

2. Twenty "clinically normal" dogs were anesthetized, given curare and artificial respiration with the E and J Resuscitator, a suck (—9 mm. of mercury) and blow (+14 mm. of mercury) type, for one to three hours. The animals were then permitted to recover and were killed three days later and the lungs examined by a method permitting the discovery of pneumothorax and interstitial or mediastinal emphysema. Two dogs (8 per cent) died of bronchopneumonia within seventy-eight hours; since gray hepatization was present, the bronchopneumonia must have been present at the time of the experiment. Four (16 per cent) of the dogs had minor pathologic changes in their lungs. In the remainder (76 per cent) the lungs were normal.



Illustrating that an animal in coma (anesthetized) but breathing naturally may or may not follow the rhythm of a mechanical resuscitator of the "suck and blow" type. In the first section of the tracing the animal was breathing naturally. In the second section the E and J Resuscitator was ventilating the animal (note that the animal is "following" the resuscitator). In the third section the animal is not "following" the resuscitator. In the fourth section the animal is apparently not "following" the resuscitator, but when the resuscitator is turned off and the animal allowed to inhale 100 per cent oxygen without pressure naturally the natural respiration is irregular, a condition which continues after the resuscitator is turned on again. An irregularity of the respiratory excursion may occur during natural breathing in anesthetized or comatose animals.

3. There is no evidence in this study that artificial respiration of the suck and blow type (+14 mm. of mercury and —9 mm. of mercury) caused lung damage.

4. In 5 anesthetized dogs given artificial respiration with the E and J Resuscitator for one hour in the presence of natural breathing no evidence of emphysema was found.

5. The rate of return of blood pressure to the control level after the administration of curare was studied when dogs were given artificial respiration by various mechanical methods for one to three hours. The E and J Resuscitator was used on 20 dogs, the General Electric intermittent positive pressure Pneumolator on 7, the B intermittent positive pressure device on 8 and the Drinker Respirator on 7. No difference was found.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

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SATURDAY, DECEMBER 29, 1945

INFECTION AND REINFECTION IN TUBERCULOSIS

Since the work of Parrot, Küss, Ghon, Ranke and many others there has remained little doubt that the origin of tuberculous disease in infants and young children is almost exclusively the first contact with tubercle bacilli. Furthermore, Koch, Romer, Krause and many others found that it was difficult to superinfect an active infection in experimental animals. By analogy an exogenous reinfection in children before 3 years of age probably does not occur often if at all, because the lesions do not have time to heal within that time.

At one time most adult tuberculosis was thought to arise from these early infections. Late in the last century Behring made the often quoted statement that fatal tuberculosis was the "last verse of the song begun in the cradle." Not all were in agreement with Behring's hypothesis, however, because Nägeli, Orth and others had been reporting the presence of healed calcified tuberculous lesions on necropsy in practically every adult. As late as 1926 Schürmann [1] found that almost all adults possessed some tuberculous lesions—mostly healed and calcified. The tuberculin reaction supported the finding of a nearly universal infection in the large centers of population. The use of tuberculin by Hamburger and Monti,[2] Armstrong,[3] Veeder and Johnston [4] and others indicated that most of the people in the large centers of population were infected by the time they reached adult life. With apparently so many lesions existing in the world, it seemed probable that healing must take place or every one would be ill or have latent disease. Although many early experiments reported a high percentage of tubercle bacilli in calcified tuberculous lesions, Rabinowitch [5] and Aronson and Opie [6] found a much lower percentage of positive lesions. More recent studies [7] have demonstrated that only a small percentage of the lesions contain living tubercle bacilli. Practically none of the "petrified" or "ossified" lesions contain living bacilli. The conclusion was correct that most calcified lesions were probably healed.

The next logical question was whether these healed infections protect against subsequent reinfections, as animal experiments seemed to indicate. Many felt that there was real immunity conferred by a healed infection. Even if all the lesions were healed and afforded complete protection (as they surely do not) not all persons were infected in early life. Bushnell,[8] Borrel [9] and others showed that aboriginal people presented less evidence of infection as they were remote from civilization. A similar variation also existed in certain civilized communities. In rural Minnesota as early as 1924 Slater [10] found that only 10 per cent of high school children had positive tuberculin (Pirquet) reactions by the time of graduation. Scheel [11] in Norway, Myers [12] in Minneapolis and Taylor [13] in Iowa supported Slater's observations.

Not only did tuberculosis vary from place to place, but there was increasing evidence that the incidence of the disease was rapidly decreasing. The best confirmation of this statement, besides the decrease in mortality, was the work of Chadwick and Zacks [14] in Massachusetts, who found the incidence of the disease in 1930 to be about half that found by Armstrong in 1917. Furthermore, Chadwick and Pope [15] found 28 per cent positive tuberculin reactions in grade school children in 1924 and only 14 per cent positive in 1939. It is evident that there is a vast difference in the tuberculosis infection rate, total infection, morbidity and mortality in different parts of the world, even in different parts of every country, and the incidence is changing all the time. Except in war and famine, at least in the more civilized countries, the rate is decreasing.

Observations reported on special groups of patients should not be deceptive. For example, the fact that

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2. Hamburger and Monti: Die Tuberculosehäufigkeit im Kindes alter, München. med. Wchnschr. **56:** 449, 1909.
3. Armstrong: The Framingham Demonstration: The von Pirquet Survey, Bull. Nat. Tuberc. A., 1919.
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14. Chadwick, H. D., and Zacks, D.: Incidence of Tuberculous Infection in School Children, Am. Rev. Tuberc. **22:** 626 (Dec.) 1930.
15. Chadwick, H. D., and Pope, A. S.: The Modern Attack on Tuberculosis, Am. J. M. Sc. **189:** 253 (Feb.) 1935.

practically all infants and young children who die of tuberculosis do so as a result of the primary infection does not indicate that a larger number of childhood infections survive, as shown by Bernard [16] and by Kjer-Petersen and Ostenfeld.[17] The majority of these survivors live to become reinfected later. The same may be said regarding the question of endogenous reinfection, as reported by Sweany,[18] Pagel [19] and others. Although young adults in particular show a preponderance of endogenous reinfections, it must be remembered that many more infections heal than result in death and in healing become subject to later reinfections.

The percentage of persons who heal compared to those who die is not known, largely because there are so many variable factors, but there is little doubt that for every person dying of tuberculosis many more survive, and the proportion of survivors to those infected increases in the older age groups. Exogenous reinfection, therefore, increases in importance with age. As the percentage of uninfected persons increases, that is, as adult life and old age are attained without a first infection, reinfections become less and less significant. The time has by no means come, however, to disregard reinfection on that account.

Theoretically, then, infection and reinfection can occur at any time if the dosage and virulence of the bacilli are high enough and if the host has a low resistance as the result of a susceptible age, sex or race or if there has been a loss of resistance, as in old age. This holds true also in any case in which the first infection was slight and a long interval of time elapsed between the first infection and the reinfection. A patient with active tuberculosis expels millions of bacilli daily, may infect any one, and therefore is a menace not only to the uninfected but also to those in the older age group who have healed infections.

Old age tuberculosis has no doubt been largely overlooked in the past except at necropsy. Recently there have been a number of reports on tuberculosis in persons in the older age groups—probably not because the disease is more prevalent but because better diagnoses are being made.

Probably well over half of the fatal cases of tuberculosis still arise endogenously from the first infection, and as the infection rate decreases the importance of reinfection decreases. There is, however, a large percentage of clinically significant and even fatal disease in which the infections are due to exogenous reinfections, which may be few in the young, many in early middle life and quite prevalent in the old.

16. Bernard, Leon: Les débuts et les arrêts de la tuberculose pulmonaire, Paris, Masson et Cie, 1931.
17. Kjer-Petersen, R., and Ostenfeld, J.: Positive Pirquet Test in First Three Years of Life, Ugesk. f. læger **92**: 865 (Sept. 11) 1930.
18. Sweany, H. C.: Recent Studies Bearing on Tuberculous Infection and Reinfection, Acta tuberc. Scandinav. **13**: 67, 1939.
19. Pagel, W.: On the Endogenous Origin of Early Pulmonary Tuberculosis, Am. J. M. Sc. **189**: 253 (Feb.) 1935.

ANTITOXIC ACTION OF PENICILLIN

Boor and Miller [1] of the Kuppenheimer Foundation, University of Chicago, have recently demonstrated that penicillin has a synergic neutralizing action on certain endotoxins. Endotoxins were prepared from six strains of type I and one strain of type II meningococcus. The concentration of each preparation was so adjusted that 0.3 to 0.5 cc. injected intraperitoneally killed at least 4 of 5 mice within thirty hours. Among 179 untreated control mice thus injected 159 died within thirty hours, a mortality of 89 per cent. In comparison 74 mice were injected intraperitoneally with the same doses mixed with 1,000 or more units of penicillin. Of these 59 died within the prescribed time limit, a mortality of 80 per cent. Boor concluded from these percentages that penicillin acting in vitro on meningococcus endotoxin does not reduce its toxicity to any significant degree. This conclusion accords with that previously reported by Neter and Will,[2] who found that tetanus toxin mixed with sodium penicillin was as toxic as tetanus toxin alone.

In a second series of experiments Boor injected the same amounts of endotoxin intraperitoneally into mice which were treated by a series of subcutaneous injections with penicillin. Of 373 mice thus treated 122 died, a mortality of but 33 per cent.

The antitoxic effect was even more apparent in several groups of mice injected with smaller doses of endotoxin. One group of 24 mice was injected with but 0.25 cc. of standard endotoxin. The control mortality was 100 per cent, contrasted with but 20 per cent mortality among those given subcutaneous penicillin therapy. Experiments were also done on rabbits. In rabbits the endotoxin was injected intravenously and penicillin given subcutaneously in doses of about 10,000 units every few hours. All untreated control rabbits died within twenty-four hours (mortality 100 per cent). Among 16 treated rabbits 14 survived and 2 died at thirty-six and forty-five hours respectively, a mortality of but 12.5 per cent.

These experiments confirmed the previous conclusion that penicillin repeatedly administered subcutaneously is able to exert a considerable degree of protection against the lethal effects of meningococcus endotoxin. This protective action is almost completely destroyed when penicillin is inactivated with penicillinase.

The Chicago investigators have not yet proposed a theory to account for this presumptive synergistic antitoxic effect of penicillin, a new phenomenon of clinical promise.

1. Boor, A. K., and Miller, C. P.: Effect of Penicillin on Lethal Action of Meningococcal Endotoxin in Experimental Animals, Science **102**: 427 (Oct. 26) 1945.
2. Neter, Erwin, and Will, Dessie: Effects of Penicillin, Clavacin and Streptomycin on Tetanus Toxin, J. Bact. **48**: 261 (Aug.) 1944.

Current Comment

FIBROSITIS OF THE BACK

Copeman and Ackerman [1] reported in 1944 a clinical study of "fibrositis" of the back in which the pain was studied clinically and the occurrence of trigger points confirmed. The exact situation in which these occurred in a large number of patients was charted and the pain pattern of definite shape obtained. The dissection of 14 bodies of persons dying from various causes, excluding those of fibrositis, established the existence of a basic fat pattern. This fat pattern was observed to correspond exactly in shape and situation with the pain pattern already charted. Lesions suggestive of inflammatory reaction were not found in any deep fibrous tissue, but certain abnormalities affecting the lobules of the fat pattern were found in several instances. The abnormality which seemed most likely to have clinical significance was the herniation of certain fat lobules through weaknesses or actual deficiencies in the walls of their investing fibrous tissue. A series of 10 biopsies of selected patients suffering from fibrositis were reported in full. Although the authors think that the microscopic lesions described in the removed fat may be causative of the pain, they believe that its basic origin lies in an increase of tension in the fat lobules affected where they are invested by a nondistensible fibrous membrane. The anatomic lesions found were probably a later result of this condition where it persists. Further clinical support to the concept of herniation of fascial fat as a cause of back pain is offered by Herz,[2] who reports a striking relief of pain in 6 patients suffering from excruciating pain in the back. In all of his cases there was a history of trauma due to strain, which had initiated the pain. Pain in the back was extremely severe in all and in some instances was referred down one leg. In all cases a definite mass was palpable which was extremely tender, and pressure on it intensified the pain. Injection of these masses with procaine solution provided temporary relief in all instances. In these 6 cases, fairly wide dissection of fat in the painful area has relieved the pain whether or not the herniation could be clearly delineated. Mylechreest,[3] who dissected 12 backs of bodies post mortem, furnishes additional information. In several of the bodies a herniation of fat from one layer of fascia to another was found. In 12 biopsies on selected patients suffering from fibrositis of the back, fatty lobules under tension or fatty hernias were discovered. Their removal resulted in cure in most cases or striking relief in those not completely cured. The fat hernia, according to Mylechreest, may be congenital in origin, where a mass of fat passes through one or more layers of fascia through well defined gaps, or acquired, the result of a mechanical or postural factor. The pain of "fibrositis" may be due to pathologic changes found in these areas, namely edema of the fatty tissue, hemorrhage, congestion or torsion of the pedicle of the prolapsed lobule. Herz suggests that the operation for the release or removal of prolapsed herniation of fascial fat is not a panacea for all types of low back pain. However, if the possibility of such a lesion is recognized and a careful examination is made for palpable masses which may prove to be trigger points of pain, numerous patients may by this procedure be relieved of a condition that has been incapacitating, sometimes for many years.

1. Copeman, W. S. C., and Ackerman, W. L.: "Fibrositis" of the Back, Quart. J. Med. **13**: 37 (April-July) 1944.
2. Herz, Ralph: Herniation of Fascial Fat as a Cause of Low Back Pain, J. A. M. A. **128**: 921 (July 28) 1945.
3. Mylechreest, W. H.: Investigation into the Etiology and Pathology of Fibrositis of the Back, Ann. Rheumat. Dis. **4**: 77 (June) 1945.

CONFERENCE ON BARBITURATES

The Committee on Legislation of the American Pharmaceutical Association recently called a conference to study the promiscuous distribution of the barbituric acid compounds. The meeting was attended by representatives from the American Pharmaceutical Association, American Medical Association, Food and Drug Administration, U. S. Public Health Service, U. S. Bureau of Narcotics, American Dental Association and other groups and agencies. Discussion was held on barbiturate regulation, barbiturate habituation and tolerance and the medical, pharmaceutical and legal aspects of the problem. At this time fourteen states and the District of Columbia have no law regulating the sale and distribution of barbiturates. The laws in the other states are not uniform and have been subjected to much abuse. Today many people are aware of the action of this group of drugs; accidental deaths, suicides and ill health directly attributable to the effect of the drugs are on the increase. Careless prescribing, direct sales to the consumer and "bootleg" distribution have aided those who are determined to get the drug. Some have proposed the adoption of a federal law which would regulate the distribution of barbiturates much as opium derivatives are now controlled. More are in favor of control on the state level, particularly if a uniform state act can be formulated and adopted. The problem needs much study. Further study should develop a satisfactory solution.

PROMIN IN LEPROSY

Faget and Pogge [1] at the National Leprosarium in Carville, La., report clinical improvement in 137 patients with lepromatous and mixed types of leprosy treated with promin. The drug acts slowly, and improvement usually becomes manifest only after six or more months of treatment. The longer the duration of promin therapy and the larger doses of the drug tolerated, the greater is the degree of improvement. Since Mycobacterium leprae cannot be cultivated on artificial mediums nor the human disease reproduced in laboratory animals, bacteriostatic or bactericidal action of promin is difficult to prove. However, 62 patients were treated with promin for more than one year, with diminution in the number of M. leprae organisms in the lesions in at least 40 per cent. Research may produce a faster acting, more specific, drug for the mycobacterial diseases, but promin is considered the best experimental treatment thus far tested at the National Leprosarium.

1. Faget, G. H., and Pogge, R. C.: The Therapeutic Effect of Promin in Leprosy, Pub. Health Rep. **60**: 1165 (Oct. 5) 1945.

MEDICINE AND THE WAR

ARMY

DISTINGUISHED SERVICE MEDAL

Major General George F. Lull

Major Gen. George F. Lull, Deputy Surgeon General of the Army, was recently awarded the Distinguished Service Medal for "exceptionally meritorious conduct in the performance of outstanding services in the Office of the Surgeon General from June 1940 to August 1945." The citation accompanying the award stated that "in his capacity as chief of personnel service he was responsible for developing plans to augment the various officers' corps and the enlisted and civilian personnel of the Medical Department during the nation's first total mobilization of medical manpower. As Deputy Surgeon General he was largely responsible for establishing policies and directing studies which resulted in many outstanding medical achievements such as the advancement in preventive health measures, the remarkably low incidence of disease and the low mortality from both disease and battle wounds. General Lull's skilful discharge of difficult duties and his devotion to the mission of the medical department contributed in important degree to the success of the Army's unprecedented medical program." General Lull graduated from Jefferson Medical College of Philadelphia in 1909 and was commissioned in the Army Medical Reserve in 1912.

Major General Shelley U. Marietta

Major Gen. Shelley U. Marietta, commanding general of Walter Reed General Hospital, Washington, D. C., was also the recipient of the Distinguished Service Medal. The citation accompanying the award stated that as commanding general of Walter Reed General Hospital as well as of the Army Medical Center and a commandant of the Medical Department Professional Service Schools from December 1939 to August 1945 he "displayed outstanding leadership and administrative and professional ability of a high order in discharging his highly responsible duties. He organized and trained medical department units and personnel in medicomilitary activities while at the same time carrying out his responsibilities for the proper performance of the many-times expanded missions of the Army Medical School, the Enlisted Technicians School, the Army Dental School, the Army Veterinary School and the Walter Reed General Hospital. Under his guidance the armywide needs for blood plasma and both the Army and Navy needs for immunizing biologicals were always fulfilled. He helped pioneer numerous innovations for the care and treatment of the sick. General Marietta's contribution to the war effort, made possible by the diligent application of his exceptional talents, was an important one and reflects highest credit on himself and the military service." General Marietta graduated from the University of Illinois College of Medicine, Chicago, in 1909 and entered the service May 1, 1912.

Brigadier General James S. Simmons

Brig. Gen. James S. Simmons, Washington, D. C., was recently the recipient of the Distinguished Service Medal. The citation stated that "the service he organized and developed carried out a worldwide program of military and civil public health which secured both immediate and enduring benefits by reducing hazards to the health of the troops, civilians engaged in essential war work, and refugees and displaced persons. By applying the best available knowledge and fostering research, he developed and extended new information on the causes and prevention of communicable diseases, on the improvement of nutrition for soldiers and on the reduction or elimination of the health hazards of mechanized warfare and industrial occupations. His teachings, influence of personality and adherence to ideals inspired and established a positive concept of health for troops and civilians. With extraordinary foresight he was in advance of events, devising measures for health protection before critical needs arose, and with dynamic energy he overcame severe difficulties. By protecting the health of the Army and conserving the health of the nation, General Simmons contributed to winning the war." General Simmons graduated from the University of Pennsylvania School of Medicine, Philadelphia, in 1915 and entered the service Feb. 10, 1917.

Brigadier General Raymond W. Bliss

Brig. Gen. Raymond W. Bliss, assistant Surgeon General of the Army, received the Distinguished Service Medal for "exceptionally meritorious conduct in the performance of outstanding service from June 29, 1943 to Aug. 31, 1945." The citation further stated that "General Bliss was responsible for the development and supervision of the medical plan in overseas theaters and in the zone of interior, which through its excellence provided the finest medical service in the history of the Army and resulted in the lowest morbidity and mortality experience. Through personnel planning and control, through specialization of facilities, through the furtherance of such new developments as air evacuation and convalescent hospitals, General Bliss contributed uniquely to the treatment and welfare of the sick and wounded and the accomplishment of the medical mission." General Bliss graduated from Harvard Medical School, Boston, in 1910 and was commissioned in the Army Medical Reserve Corps in 1911.

ARMY MEDICAL LIBRARY HONORARY CONSULTANTS MEET

The second annual meeting of Honorary Consultants to the Army Medical Library was held recently in Cleveland for the purpose of electing officers to the association. The following officers were elected: Dr. John F. Fulton, president; Dr. Chauncey D. Leake, vice president; Col. Harold W. Jones, secretary-treasurer. Major Gen. George F. Lull was elected on the executive committee.

ARMY AWARDS AND COMMENDATIONS

Colonel William S. Middleton

The Legion of Merit was recently awarded to Col. William S. Middleton, formerly of Madison, Wis., for "outstanding services as chief consultant in medicine, Professional Services Division, Office of the Chief Surgeon, in which capacity he established and directed the consultative services in medicine in the European theater." Dr. Middleton graduated from the University of Pennsylvania School of Medicine, Philadelphia, in 1911 and entered the service April 27, 1942.

Colonel Paul G. Hansen

Col. Paul G. Hansen, formerly of North Olmsted, Ohio, and now attached to a medical detachment in China, was recently awarded the People's Hero Medal by the Chinese government. For the last two years he has been in charge of hospitals in central and western China. Some time ago he was awarded the Bronze Star. Dr. Hansen graduated from St. Louis University School of Medicine in 1933 and entered the service April 22, 1940.

Captain Herman Beigelman

Capt. Herman Beigelman, formerly of Boston, was recently awarded the Bronze Star for meritorious achievement in connection with military operations in Italy. Dr. Beigelman graduated from Tufts College Medical School, Boston, in 1937 and entered the service Nov. 20, 1942.

MISCELLANEOUS

AWARD OF UNITED STATES OF AMERICA TYPHUS COMMISSION MEDAL

Col. Richard T. Arnest, formerly of Fort Benning, Georgia, was recently awarded the United States of America Typhus Commission Medal. Accompanying the award was a citation "for meritorious service in connection with the work of the United States of America Typhus Commission during the outbreak of epidemic typhus fever in southern Italy, particularly during the period from Dec. 20, 1943 to Feb. 20, 1944. Colonel Arnest from his position as surgeon, Peninsular Base Command, made available to the commission personnel and facilities. In addition he carried out all responsibility for applying measures necessary to the prevention of the occurrence of typhus fever in United States Army forces in the Naples area. His contribution not only assisted typhus control among civilians but also safeguarded the health of troops." Dr. Arnest graduated from the University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, in 1916 and entered the service July 1, 1920.

Lieut. Col. Wilson C. Williams, formerly of Nashville, Tenn., was recently presented the United States of America Typhus Commission Medal. According to the citation accompanying the award, "as deputy director of public health subsection, Allied Control Commission, and director of the typhus control branch of that subsection, he rendered extremely valuable services in planning for antityphus supplies, in training personnel and in assisting in the coordination of activities for the control of typhus during the outbreak of this disease in Naples and southern Italy during the period from December 1943 to February 1944. After the withdrawal of the United States of American Typhus Commission from control work at Naples, Colonel Williams continued an effective campaign against typhus in southern Italy." Dr. Williams graduated from Vanderbilt University School of Medicine in 1925 and entered the service Aug. 18, 1943.

A BRITISH TRIBUTE TO THE AMERICAN PHYSICIAN

In the London *Lancet* for October 20, page 507, an anonymous writer ventures a few reminiscent thoughts relative to the association of the British physicians with their American colleagues during the period of the war. It is a charming statement in which every medical reader should take pleasure. The statement follows:

They came, at first a few and then a drove, the Æsculapiads of the New World to succour wartorn Europe. Khaki-clad and with British names and customs they shared our clubs, our meetings, and our firesides. Up sprang their hospitals almost overnight: clean wards and messrooms, well-ordered stores, theatres, and pharmacies. In their laboratories were shiny benches, virgin glassware, and crystal-clear solutions. Smart white-gaitered sentries "checked in" from their white-painted control offices. In the motor pool stood strange high monsters of the road, regimented for war and errands of mercy. There were libraries, cinemas, and baseball grounds, as well as cosy bars with lantern lights, iced fruit cocktails, and exotic wall-paintings. They shared out parcels from home in their warm Nissen huts and put up on the stark concave walls photographs of their far-away families. Off plain deal tables they ate prefabricated steaks and Maryland chickens with apricot jam. They poured out sweetcorn from cans and treated us to large fruity ices and hot chocolate sauce. There was pride in their handicraft, as their chapels showed, for only their best would do. Everywhere efficiency and quality, with sufficient of everything for the job in hand. Then came the convoys and their jobs began. The sterilisers steamed, the X-ray plants hummed, and the centrifuges roared. The purple dressing-gowns of the convalescents made their welcome appearance as blood, penicillin, and faith in the future speeded them back to their units.

The months passed and we got to know them—colonels, doctors, nurses, and enlisted men. Many were the meetings and the consultations. They treated our men and we treated theirs. They came to learn, youths of a young nation, but they taught us much. They transformed our sleepy towns into energetic schools of medicine. In leisure hours they swarmed our country lanes on bicycles and in rare bouts of sunshine played with our children on the greens, giving them candies and peanuts. Some married our daughters and our sons. They loved our ancient churches and studied the flowers in our meadows. They had aunts to visit, ancestral homes to photograph, and souvenirs to seek. The aroma of strange tobacco pervaded our streets. Hospitable and witty, they were the possessors of good manners and good teeth. They tolerated good-humouredly our lack of central heating and culinary understanding, and enjoyed above all the humble cup of tea in the family circle. They told us of their homes and we wrote to their families. Strange world that they who speak our language, share our jokes and ancestry, and fight the same battle against disease should be reckoned foreigners to us and we foreigners to them. Citizens of the same world, we must needs be in separate sovereign States and perhaps willy nilly be required to serve separate interests.

They went as quickly as they came, for the lightning has severed the chains of bondage. The hospital trains have ceased to run. The wounded are healed and have left for home. The hospital cities are dismantled and the men in the Nissen huts have packed their books, souvenirs, and photos and filed off to the ports in their fantastic trucks. The bulldozers have levelled the last dump of empty cans and all is still. There is dust on the empty shelves of the pharmacy, the little trim gardens are overgrown, and the white sentry-box is empty. Now, only the miniature house is inhabited which the mess sergeants built for the tomtits. The abandoned huts sadden our eyes as we recall friendships fashioned in the hinterland of war. The stimulus of their presence is now a memory, for three thousand miles, and a disordered world economy, separates us from them, truly our cousins. And our lazy old town has lapsed into dreams again.

But they will write and remember us. And we shall not forget them.

PS.—I wish we had a cosy bar in our hospital.

UNRRA NEWS

Successful Experiment by Sweden

Dr. R. L. Coigny, UNRRA's medical chief for displaced persons in Europe, recently reported the success of an experiment by Sweden in cooperation with UNRRA in building up children starved by the Nazis. Though not a member of the United Nations, Sweden has accepted through UNRRA a total of 31,000 slave laborers and war victims who were in especially bad health when liberated from Nazi prison camps last spring. The Swedes provided the refugees with a diet of 4,800 calories daily, although the normal ration for Swedish citizens is only 3,800. The enjoyment of plenty following the privation and suffering experienced by these people under the Nazis has not only rehabilitated them physically at an unusual rate but has also helped to restore their mental health with remarkable rapidity. The displaced persons are housed in 150 centers of different types, some being large converted country houses, others gaily painted bungalows, all located in beautiful surroundings.

Although Sweden has borne the entire cost of their care, UNRRA is cooperating on plans for their repatriation. Originally turned over to UNRRA in Germany by the military forces after their liberation, the displaced persons were registered by UNRRA workers at Lubeck and transported across the Baltic aboard Swedish ships. The transfer took place between June 25 and July 27. Sweden has placed no specific limit on the time they may remain in hospitals or convalescent centers.

Medical Supplies to Italy

The United Nations Relief and Rehabilitation Administration, Washington, D. C., recently made a shipment of 900 tons of medical supplies for distribution through Italian government authorities, and more are on the way. Mr. Rickard K. Myers, assistant chief of UNRRA's Medical and Sanitation Supplies Division, is in Rome to aid in supervising the distribution. The shipment was made up of 23,000 separate cases of the vital medicines and equipment. Already 2,800 cases have been shipped to provinces most in need of them, including Sicily, Lucania, Campania, Abruzzi and Lazio, and they are being distributed to hospitals and health centers for immediate use. UNRRA already has provided 100 ambulances in Italy, which it obtained from military surpluses in Europe.

PHYSICIANS SEPARATED FROM SERVICE

ARMY MEDICAL CORPS OFFICERS RECOMMENDED FOR/OR RELIEVED FROM ACTIVE DUTY

Alabama

Burns, Charles R. D., Capt., Alabama City.
Patton, Thomas H. Jr., Capt., 12 Audubon Pl., Tuscaloosa.
Roberts, Rufus W. Jr., Capt., % R. W. Roberts, N. Y. Life Ins. Co., Frank Nelson Bldg., Birmingham.
Snelling, David B., Major, Eutaw.
Spruell, William H., Lt. Col., Russellville.

Arizona

Creighton, Carrol C., Capt., 18 W. Aspen, Flagstaff.

Arkansas

Johnson, Julius D., Major, P. O. Box 566, Fort Smith.
King, Joe W., Capt., 310 Cherry St., Helena.
Parker, William M., Capt., DeVall's Bluff.
Walls, James M., Major, Blytheville.

California

Abbett, Arthur L., 1st Lt., 5708 Florence Ave., Oakland.
Cromenwett, Paul H., 1st Lt., 1729 Cornell Dr., Alameda.
Dresser, James T., Capt., 1621 G St., Bakersfield.
Esnard, John E., Capt., 1608 S. Durango Ave., Los Angeles.
Friedman, Meyer, Capt., 101 Point Lobos Ave., San Francisco.
Hodgdon, Frank W., Major, 1110 Arden Rd., Pasadena.
Jaekle, Russell F., Major, 450 Sutter St., San Francisco.
Jeans, Virgil E., Major, 701 Patterson Bldg., Fresno.
Kaiser, Joseph E., Capt., 818 16th St., Santa Monica.
Karr, Martin, Capt., 3150 Chelsea St., San Mateo.
Kesterson, Robert L., Major, % Dr. W. J. Kesterson, U. S. Vet. Fac., Livermore.
Kladunde, Paul W., Capt., 105 18th Ave., San Francisco.
Klein, Russell R., Capt., 723 12th Ave., San Francisco.
Lehman, H. O., Capt., 69th Coast Artillery (AA), San Diego.
Littleton, William H., Capt., 4335 Illinois St., Fresno.
Minna, John B., Major, 3790 Park Blvd., San Diego.
Mitchell, William J., Lt. Col., Alhambra.
Munal, Harold D. Jr., Capt., 1212 Shatto St., Los Angeles.
Newman, Irving G., Capt., 145 S. Reeves Dr., Beverly Hills.
Phillips, Albert D. Jr., Lt. Col., 3310 H St., Sacramento.
Price, J. B. Melville, Lt. Col., 921 Louise St., Santa Ana.
Richey, Tim V., Capt., Box 73, El Cajon.
Russell, Carroll A., Capt., 2737 22d St., San Francisco.
Shapiro, Newton H., Capt., 30 Casa Way, San Francisco.
Smith, Ernest T., 1st Lt., Glendale.
Wood, George A., Lt. Col., 1437 Hamilton Ave., Palo Alto.

Colorado

Friedman, Gerald H., Capt., 1420 Logan, Denver.
Lord, George H., Capt., 1460 Chester St., Aurora.
Stander, Thomas R., Lt. Col., 735 Steele St., Denver.

Connecticut

Chiota, Joseph A., Capt., 147 Terry Pl., Bridgeport.
Glike, Frederick P., Capt., 106 Columbus Ave., Meriden.
Krosnick, Morris Y., Capt., 105 Canner St., New Haven.

Delaware

Stern, Oscar N., Major, 1016 Rodney St., Wilmington.

District of Columbia

Barnes, Earl B., Capt., Gallinger Municipal Hosp., Washington.
Chase, Morris, Lt. Col., 3929 13th St. N.W., Washington.
Difrancesco, Vincent J., Capt., 3356 Pa. Ave. S.E., Washington.
Hyman, Daniel, Capt., 5603 16th St. N.W., Washington.
Raffel, William, Capt., 4815 Georgia Ave. N.W., Washington.
Schreiber, John O., Major, Garfield Mem. Hosp., Washington.
Williamson, Ben K., Major, 1915 Kalorama Rd. N.W., Washington.

Florida

Bell, Francis E., Major, 415 Emmett St., Palatka.
Burch, John E., Capt., 2024 S.W. 19th St., Miami.
Feaster, Orin O., Lt. Col., 166 4th Ave. N.E., St. Petersburg.
Gable, Nonie W., Col., 706 Power & Light Bldg., St. Petersburg.
Levy, Morton, Capt., 521 Revere St., Orlando.
Weinstein, Abraham S., Capt., Box 527, Vets. Hosp., Lake City.

Georgia

Billinghurst, George A., Major, State Hosp., Milledgeville.
Garner, John P., Capt., 607 Med. Arts Bldg., Atlanta.
Holden, Howard T., Major, Rabun Gap.
Mann, Frank R. Jr., Major, McRae.

Idaho

Kaar, Richard C., Capt., Box 228, Burley.

Illinois

Abrahams, Benjamin, Capt., 7025 Sheridan St., Chicago.
Armalavage, Leon J., Major, 1819 Polk St., Chicago.
Aronoff, Joseph, Capt., Morton.
Baker, Philip M., Capt., 7802 Colfax Ave., Chicago.
Baylor, John E., Capt., 621 Waveland Ave., Chicago.
Benedek, Tibor, Lt. Col., 1706 E. 56th St., Chicago.
Carney, Thomas B., Major, 611 S. Tremont St., Kewanee.
Clark, John S. Jr., Capt., 1006 W. Stephenson, Freeport.
Cohan, Albert, Capt., 2724 Crystal St., Chicago.
Conner, James A., Major, 8913 Blackstone Ave., Chicago.
Cozad, Delos R., Capt., 500 Diversey Parkway, Chicago.
Cunningham, Glenn D., Major, 915 30th St., Rock Island.
Dean, Robert K., Capt., 400 W. Forrest Hill Ave., Peoria.
Fahlstrom, Stanley C. W., Col., 30 N. Michigan Ave., Chicago.
Foley, Charles J., Major, 535 Madison St., Waukegan.
Fraerman, Samuel H., 1st Lt., 628 Sheridan Rd., Chicago.
Garvy, Andrew C. Jr., Capt., 5842 Wayne Ave., Chicago.
Giltner, Otis B., Major, Sheffield.
Hammatt, Harold W., Capt., 547 W. Jackson Blvd., Chicago.
Harris, Garford R., Capt., 6549 S. Green St., Chicago.
Jana, Edward C., Capt., 4034 W. 21st Pl., Chicago.
Johnston, Kenneth P., Major, 27 Julian Pl., Elgin.
Kane, David S., Major, 1809 S. Central Park Ave., Chicago.
Lander, Herman B., Capt., 3922 W. 13th St., Chicago.
Leshin, Arne, Capt., 5474 Everett Ave., Chicago.
Levan, Arthur B., Capt., 3811 Pine Grove Ave., Chicago.
Logsdon, Robert E., Capt., Mount Sterling.
Lowy, Howard A., Major, 101 Elmridge Rd., Creve Coeur.
Mcewen, Kenneth W., Capt., 7660 W. Monroe St., Forest Park.
Majarakis, James D., Major, 620 E. 81st St., Chicago.
Morgan, Jack A., Major, 750 S. State St., Elgin.
Nelson, Melvin J., Capt., 4331 W. Schubert Ave., Chicago.
O'Donnell, Frederick D., Capt., 1940 Thornwood Ave., Wilmette.
Ostrowski, Fabian S., 1st Lt., 273 Blackhawk Rd., Riverside.
Parker, Francis W., Capt., 303 E. Superior St., Chicago.
Rosenblum, Earl, Lt. Col., Cook County Hosp., Chicago.
Roth, John B., Capt., 324 Liberty St., Morris.
Sandler, William S., Major, 5252 Broadway, Chicago.
Shulruff, Harry I., Major, 1833 S. Millard Ave., Chicago.
Weir, John M., Capt., 132 F. W. Riverview, Decatur.
Zurfli, Clarence J., Lt. Col., 4132 N. Kedvale Ave., Chicago.

Indiana

Amos, Robert L., Major, 924 Lincoln Ave., New Castle.
Carbone, Joseph A., Capt., 131 E. 5th Ave., Gary.
Dieckman, Herbert S., 1st Lt., R.R. 3, Evansville.
Fisher, Kenneth B., Major, 634 Associates Bldg., South Bend.
Glackman, John C. Jr., Capt., Rockport.
Heinrich, Guenter E., Capt., Box 68, Ewing.
Kolanko, Leo A., Capt., 6104 Columbia Ave., Hammond.
Miller, Roland E., Capt., 114 E. Jackson St., Plymouth.
Pasterak, A. J., Capt., U. S. Vet. Adm. Fac., Indianapolis.
Sanderson, Richard J., Capt., Westville.

Iowa

Eller, Lancelot W., 1st Lt., Kanawha.
Heitzman, Paul O., Capt., 618 Med. Arts Bldg., Burlington.
Knott, Robert C., Major, 620 Davidson Bldg., Sioux City.
Long, Draper L., Capt., 1104 W. State, Mason City.
McMeans, Thomas W., Capt., 2811 Davenport Ave., Davenport.
Myers, Kermit W., Capt., Sheldon.
Riegelman, Ralph H., Major, 1201 Equitable Bldg., Des Moines.
Sharpe, Don C., Major, 1200 Main St., Dubuque.

Kansas

Brady, Charles S., 1st Lt., 837 Kansas Ave., Atchison.
Bux, Donald E., Capt., 607 S. Kansas, Columbus.
Davis, Christopher G., Major, 406 State Ave., Kansas City.
Randles, Leland P., Capt., 1001 Burke St., Fort Scott.
Saylor, Leslie L., 1st Lt., 407 Woodlawn, Topeka.
Sprong, Aaron A., Major, 112 S. Broadway, Sterling.
Williams, Ben C., Major, St. Francis.

Kentucky

Bolis, George F., Capt., 1012 Glouester Ave., Middlesboro.
Combs, Lyndon F., Capt., P. O. Box 177, Hazard.
Davis, Howell J., Lt. Col., 115 E. 4th St., Owensboro.
Fortune, Carl H., Lt. Col., Rural Route 3, Lexington.
Hahs, Robert W., Major, La Center.
Medley, Lawrence H., Capt., 2428 Allen St., Owensboro.
Monroe, Robert F., Major, 1890 Richmond Dr., Louisville.
Wells, William C., Capt., Glasgow.

PHYSICIANS SEPARATED FROM SERVICE

Louisiana

Accardo, Nick J., Capt., Box 528, Patterson.
Goldman, Allan M., Capt., 1525 Joseph St., New Orleans.
Knighton, James E., Lt. Col., 921 Ontario St., Shreveport.
Patterson, McLeod, Major, Charity Hosp. of La., New Orleans.
Pepe, John L., Major, 630 3d St., Bogalusa.
Richardson, Samuel M. Jr., 1st Lt., 116 Homer Rd., Minden.
Stander, Alvin A., Major, 125 W. Landry St., Opelousas.

Maine

Brinkman, Harry, Capt., 47 Perham St., Farmington.
Gregory, Irving F., Capt., 255 Hammond St., Bangor.
McLean, Emory A., Capt., 29 Deering St., Portland.
Steele, Charles W., Lt. Col., 472 Main St., Lewiston.

Maryland

Graham, Richard W. Jr., Lt. Col., 3702 Greenway, Baltimore.
Greene, Lawrence W., Capt., 53 Calvert St., Annapolis.
Hamburger, Louis P. Jr., Capt., 100 McHenry Rd., Pikesville.
Linhardt, E. G., Capt., 3 Chesapeake Ave., Eastport, Annapolis.
Owen, John K., Major, Hosp. for Women, Baltimore.
Rosenfeld, Morris, Capt., 5805 Clover Rd., Baltimore.
Whedbee, James S. Jr., Capt., 911 W. Lake Ave., Baltimore.

Massachusetts

Ascher, David S., Major, 1657 Commonwealth Ave., Boston.
Barton, Walter E., Lt. Col., Worcester State Hosp., Worcester.
Des Chenes, Albert A., Capt., 72 Velisle St., Fitchburg.
Dosik, Max, Capt., 24 St. Germain St., Boston.
Doyle, Richard J., Capt., 88½ Federal St., Salem.
Echlov, Theodore, Capt., 65 Bainbridge St., Roxbury.
Espinoza, David V., Capt., 628 Dudley St., Dorchester.
Frazee, John R., Capt., 25 Allendale Way, Dedham.
Jacobs, Herbert, Capt., 148 Woodlawn St., Springfield.
Jacobs, Maurice, Capt., 48 Mt. Everett St., Dorchester.
Kanski, Francis A., Capt., Mercy Hosp., Springfield.
Long, Norman G., Major, 14 Luce St., Lowell.
Maloney, John D., Lt. Col., Box C, Waverley.
Milden, Forrest, Capt., 42 Newcomb St., Haverhill.
Picariello, Americo, Capt., 295 Main St., Medford.
Silverman, Nathan, Capt., 301 Essex St., Lawrence.
Weiss, Samuel, Capt., 217 Harvard St., Dorchester.
Wilson, Franklin L., Capt., 35 Day Ave., Westfield.

Michigan

Agate, George H., Major, 2307 Vinewood Blvd., Ann Arbor.
Bryce, James W., Capt., 1814 Stone St., Saginaw.
Byrn, Robert W., Capt., 1078 Ferdon Rd., Ann Arbor.
Fenech, Harold B., Major, 10 Peterboro St., Detroit.
Giddings, Ralph R., Capt., Bloomingdale.
Huffman, Elston R., Capt., 135 Greendale West, Detroit.
Hyman, Samuel J., Capt., 27371 Michigan Ave., Inkster.
Kahn, Edgar A., Lt. Col., Univ. Hosp., Ann Arbor.
Kimberlin, Kenneth K. Jr., 1st Lt., 5005 Bedford Rd., Detroit.
Lynch, Vincent A., Capt., 1618 16th St., Wyandotte.
McCollum, Estel B., Capt., 8925 E. Jefferson, Detroit.
Mair, Harold U., Capt., 507 Notre Dame, Grosse Pointe.
Nigro, Norman D., Capt., 15820 Dexter Blvd., Detroit.
Rom, Jack, Capt., 17579 Cherrylawn, Detroit.
Sack, Anthony G., Capt., 8315 Appoline, Detroit.
Siegel, Daniel C., 1st Lt., 2762 Glendale Ave., Detroit.
Staatz, Dumont S., Capt., 3651 SU, Univ. of Mich., Ann Arbor.
Szlachetka, Vincent E., Capt., 2691 Cortland Ave., Detroit.
Wilkes, John B., Major, Grand Rapids.

Minnesota

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Benjamin, H. G., Major, 2811 Huntington Ave., Minneapolis.
Bergh, George S., Lt. Col., 2615 Park Ave., Minneapolis.
Graves, Richard B., Capt., 311 Pine St., Red Wing.
Hauge, Erling T., Lt. Col., 1817 Oliver Ave., Minneapolis.
Kreilkamp, Bernard L., Capt., 512 4th St. S.W., Rochester.
McKibbin, John P., Capt., 430 7th St. S.W., Rochester.
Meyer, Alfred C., Capt., % Mayo Found., Rochester.
Polan, Charles G., Major, 412 S.E. Walnut St., Minneapolis.
Rumpf, Carl W., Col., 326 7th St., Faribault.
Smith, Baxter A. Jr., Major, Univ. Hosp., Minneapolis.

Missouri

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Bonanno, Louis J., Capt., St. Joseph's Hosp., Kansas City.
Hollingworth, Robert S., Major, 1052 S. Delaware, Springfield.
Klippen, Arthur J., Major, City Hosp., St. Louis.
Rosehill, David B., Capt., 906 Olive St., Room 411, St. Louis.
Tapper, Stephen M., Capt., 7541 Hiawatha, Richmond Heights.

Montana

Anderson, Murl O., Capt., Hardin.
Movius, Arthur J. Jr., Capt., 1136 N. 28th St., Billings.

Nebraska

Borgmeyer, Henry J., Capt., Dodge.
Deweese, Wilford J., Capt., 1727 Pepper Ave., Lincoln.
Krahl, John B., Major, 611 G St., David City.
Popelar, Melville V., Lt. Col., 1719 S. 16th St., Omaha.

New Hampshire

Batchelder, Walter E., Major, R.F.D. 2, Dover.
Bovaird, James E., Capt., S. Main St., Wolfeboro.
Levine, Harold D., Lt. Col., 10 S. Main St., Bristol.

New Jersey

Barolsky, Benjamin, Major, 306 Broadway, Paterson.
Brogan, Francis B., Capt., 234 E. 32d St., Paterson.
Brumbach, William K., Capt., 100 Little St., Belleville.
Buechle, Carl F., Capt., E. Orange Gen. Hosp., East Orange.
Conway, James V., Capt., 428 Elmor Ave., Elizabeth.
Einhorn, Samuel E., Major, 241 16th Ave., Newark.
Englehard, George E., Capt., 142 Seeley Ave., Arlington.
Fine, Sydney G., Capt., 868 Stuyvestant Ave., Trenton.
Gelb, Jerome, Major, 86 W. Alpine St., Newark.
Hochman, Alex, Capt., 260 Hamilton Ave., Paterson.
Husted, Gerald W., 1st Lt., 102 9th Ave., Haddon Heights.
McCreight, David W., Major, % N. J. State Hosp., Marlboro.
Nevius, John K. Jr., Capt., 921 Cedar Brook Rd., Plainfield.
Nussbaum, Herman, Major, 827 State St., Camden.
Prather, Charles G., Major, 260 Westwood Ave., Westwood.
Rinzier, Elliot, Capt., 211 Roseville Ave., Newark.
Spivack, Seymour E., Capt., 28 Trotter Lane, Elizabeth.
Wood, Earl L., Col., 225 Ballantine Parkway, Newark.
Woodman, Charles B., Capt., Holland Rd., Gladstone.

New Mexico

Koff, Salmon A., Major, Vet. Admin. Fac., Port Bayard.

New York

Abelson, Max M., Capt., Jewish Memorial Hosp., New York.
Alicandri, Bruno B., Capt., 1355 82d St., Brooklyn.
Allen, Lloyd F., Lt. Col., 44 S. Main St., Pittstord.
Alvarez, Lawrence J., Capt., 689 Franklin Ave., Brooklyn.
Andrews, Joseph T., Capt., Deaconess Hosp., Buffalo.
Benjamin, Zachary H., Capt., 41 W. 86th St., New York.
Bennett, Herman J., Capt., 41 W. 83d St., New York.
Berardi, Luke J., Major, 2 N. 8th Ave., Mount Vernon.
Berchenko, Frank, Major, 576 Eastern Parkway, Brooklyn.
Berkson, Robert S., Capt., 69 Brunswick Blvd., Buffalo.
Bivona, Charles J., Capt., 10 Arlington Pl., Newburgh.
Blair, James C., Capt., 158 W. Delevan Ave., Buffalo.
Bodkin, Edmund C., Capt., 1138 White Plains Rd., Bronx.
Bogle, John H., Major, 15 Clark St., Brooklyn.
Borne, Stanford, Capt., 233 W. 77th St., New York.
Boswell, Henry A., Capt., 112 Pinewoods Ave., Troy.
Bouton, Dale C., Capt., Welfare Hosp., New York.
Bowers, Frederick W., Major, 267 Oxford Rd., New Rochelle.
Boyd, James R., Col., 897 Sterling Pl., Brooklyn.
Brezing, Herman J., Capt., 64 Burling Lane, New Rochelle.
Brick, John J., Major, 36 W. End Ave., Binghamton.
Broadman, Sylvan A., Capt., 200 W. 70th St., New York.
Brown, Daniel N., Capt., 775 Park Ave., New York.
Brown, James E., Capt., Ogdensburg.
Burgeson, Paul A., Major, 44 Elm St., Warsaw.
Butman, Burton B., Capt., Mitchel Field, Long Island.
Cacciatore, Thomas J., Capt., 908 E. 95th St., Brooklyn.
Canter, Sidney, Capt., 2005 Vyse Ave., Bronx.
Carvalho, Milton A., Major, 9 Crary Ave., Binghamton.
Chapin, Willard J., Capt., 23 Lake St., Perry.
Chester, Benjamin J., Major, 563 Howard Ave., Brooklyn.
Cimmino, Vincent J., Capt., 324 E. 4th St., Mount Vernon.
Clemente, Frank G., Capt., 661 W. 180th St., New York.
Cliff, William M., Capt., 235 E. 22d St., New York.
Climenko, David R., Capt., 38 Euclid Ave., Delmar.
Cohen, Leon J., Capt., 1331 Rosedale Ave., Bronx.
Conway, William H., Capt., 77 Briarcliff Rd., Larchmont.
Coppola, Santo T., Capt., 60 E. Broadway, New York.
Cotton, Thomas S., Lt. Col., 18 Center St., Hornell.
Dain, Harvey J., Capt., 300 Riverside Dr., New York.
D'Arata, Edward J., Capt., 7324 11th Ave., Brooklyn.
Deutl, Rudolph F., Capt., 16 Sunnyside Rd., Scotia, Schenectady.
DeWitt, Virgil B., Capt., Chestnut St., New Paltz.
Doroszka, Vincent A., Capt., 220 E. Main St., Riverhead.
Doust, Alfred W., Major, 576 Delaware St., Syracuse.

PHYSICIANS SEPARATED FROM SERVICE

New York—Continued

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Echlin, Francis A., Major, 555 Park Ave., New York.
Eisenberg, Harold J., Major, 20 Plaza St., Brooklyn.
Elitzik, Robert C., Capt., 2907 Kingsbridge Terr., New York.
Falkenbury, Frank M., Capt., 41 Sherman Ave., Glens Falls.
Felder, Seymour L., Capt., 2 Horatio St., New York.
Findlay, Robert T., Lt. Col., 4960 Arlington Ave., Bronx.
Fine, Philip, Capt., 85-45 151st St., Jamaica.
Firestone, Frederick, Capt., 68-19 79th St., Middle Village, L. I.
Fishman, Isidore F., Major, 1018 E. 163d St., New York.
Fitzgerald, Thomas G., Lt. Col., East Greenbush.
Fleming, James S., Capt., 72 Division St., Salamanca.
Foregger, Richard, Capt., 55 W. 42d St., New York.
Fox, Lester, Capt., 2300 Kings Highway, Brooklyn.
Frank, David I., Capt., 35 E. 30th St., New York.
Freed, Isidore, Capt., 5307 Roosevelt Ave., Woodside.
French, Frank S., Capt., 1960 St. Paul St., Rochester.
Frenkel, David, 1st Lt., 86 E. 49th St., Brooklyn.
Fulton, Harry L., Capt., Bellevue Hosp., New York.
Gamble, John J., Capt., 222 Lancaster St., Albany.
Gelvin, Edward P., 1st Lt., 345 E. 77th St., New York.
Gillette, Lee, Capt., Post Graduate Hosp., New York.
Gilner, Abraham, Lt. Col., 1452 Leland Ave., Bronx.
Glassman, Samuel, Capt., 66-70 E. 111th St., Manhattan.
Gleeson, Thomas H., Major, 12 E. 86th St., New York.
Glendening, Robert S., Capt., Maine.
Gold, Barnett J., Capt., 2206 Valentine Ave., Bronx.
Goldberg, Leon H., Major, 175 N. Broadway, Nyack.
Goldberg, Nathan, 1st Lt., 1562 Ocean Ave., Brooklyn.
Golden, Maurice A., Major, 92-05 Whitney Ave., Elmhurst, L. I.
Goldstein, Kenneth, Major, 23 Claremont Ave., Buffalo.
Goldstein, Philip, Major, 2001 Morris Ave., Bronx.
Greenberg, Joseph S., Capt., 4621 15th Ave., Brooklyn.
Greenberg, Milton M., Major, 4910 15th Ave., Brooklyn.
Greenblatt, Bernard, Major, 312 E. 21st St., Brooklyn.
Greene, Barnett A., Major, 8902 Avenue A, Brooklyn.
Grob, David, Capt., 2789 Brighton St., Brooklyn.
Grogan, Richard H., Lt. Col., St. Vincent's Hosp., New York.
Grossman, Leo, Major, 1584 Lexington Ave., New York.
Guerra, Wilfred, Capt., 1635 47th St., Brooklyn.
Haas, Joseph, Col., 40 W. 86th St., New York.
Hass, George M., Major, 315 E. 68th St., New York.
Hamilton, Thomas P., Major, 111 E. 76th St., New York.
Holland, J. E., Capt., 250 Beach 139th St., Belle Harbour, L. I.
Horowitz, Max N., Capt., 2964 Williamsbridge Rd., Bronx.
Horowitz, Samuel, Capt., 1414 Shakespeare Ave., Bronx.
Hurewitz, Marvin, Capt., 4802 10th Ave., Brooklyn.
Johnson, Norman P., Major, 239 N. 2d St., Olean.
Kaplan, Gustave, Capt., 10 Monroe St., New York.
Katz, Isadore H., Capt., 713 E. Genesee St., Syracuse.
Keefe, John J., Capt., 8 Main St., Camillus.
Klingman, W. O., Lt. Col., 199 Ft. Washington Ave., New York.
Kroon, Harry C., Lt. Col., 944 Euclid Ave., Syracuse.
Kurtin, Abner, Major, 320 Central Park West, New York.
Lanzetta, Louis A., Capt., 312 E. 116th St., New York.
LeFavor, Dean H., Capt., 619 Morgan Ave., Palmyra.
Leone, Anthony J., Major, 206 Dryden Ct., Ithaca.
Levin, Jack, Major, 45 E. Main St., Freehold.
Locasto, Camillo B., Capt., 276 Linden St., Brooklyn.
London, Irving, Capt., 300 Central Park West, New York.
Long, John C. Jr., 1st Lt., 18 W. 70th St., New York.
McAllister, Ferdinand F., Capt., 622 W. 168th St., New York.
McGowan, Frank J., Col., 50 E. 77th St., New York.
McKeeby, Raymond S., Capt., 84 Main St., Binghamton.
Manning, Ephraim L., Capt., % U. S. Vet. Adm. Hosp., Bronx.
March, Edgar J. II, Capt., 1487 South Ave., Rochester.
Martin, William J. Jr., Capt., 379 Quail St., Albany.
Mastrella, Angelo A., Capt., 619 Lake Ave., Rochester.
Mauceri, Joseph C., Capt., 25A Kings Park, Long Island.
Maxwell, Edmund A., Capt., 409 State St., Carthage.
Melamed, Martin E., Major, 431 Broad St., Oneida.
Meloro, Anthony, Capt., 119 Robinson St., Binghampton.
Mermell, Lester, Capt., 78 Watkins Ave., Middletown.
Meshken, Jacob, Major, 10 Park Ave., New York.
Meyers, Reuben R., Capt., 518 Ontario St., Buffalo.
Miller, Alvah S., Major, 10 Arnold Park, Rochester.
Minden, Joseph H., Capt., 123 Morsemere Ave., Yonkers.
Mirken, Allen S., Capt., 1344 E. 10th St., Brooklyn.
Moore, Maurice J. Jr., Capt., 638 W. 168th St., New York.
Morrone, Ciacento C., Capt., 87 Dunwoodie St., Yonkers.
Mulvihill, Daniel A., Major, 175-06 Devonshire Rd., Jamaica.
Murphy, Leo J., Capt., 106 S. Clinton St., Olean.
Nogin, Herbert, Capt., 1102 Clarkson Ave., Brooklyn.
Norman, Abraham, Lt. Col., 600 E. 55th St., New York.

New York—Continued

O'Connor, John P., Capt., 128 Ocean Ave., Amityville, L. I.
Ogorzaly, Stanley F., Major, 231 N. Broadway, Yonkers.
Osborn, James W., Capt., 225 Marlborough Rd., Rochester.
Panzarella, Joseph A., Capt., 1134 Glenmore Ave., Brooklyn.
Papae, Norman, Major, 415 Ocean Parkway. Brooklyn.
Papp, Sandor D., Major, 333 Wyoming St., Buffalo.
Parks, George H., Capt., 511 E. 86th St., New York.
Pruyn, Robert M., Capt., 224 Park Ave., Yonkers.
Rancourt, Charles D., Major, 218 19th St., Watervliet.
Rau, Stanley C., Capt., 30 Richmond Ave., Amityville.
Reed, Theodore D., Lt. Col., 42 Park Ave., Amityville.
Reeves, Charles D., Capt., 201 Scott St., Newark.
Regan, Francis C., Capt., 182 Child Ave., Rochester.
Ribaudo, Charles A., Capt., 764 74th St., Brooklyn.
Richardson, Charles H. Jr., 1st Lt., Bellevue Hosp., New York.
Richter, Max, Major, 250 E. 178th St., Bronx.
Rider, T. L., Capt., Stop 20, Albany Schenectady W., Albany.
Rifkin, Samuel, Capt., 1291 Eastern Parkway, Brooklyn.
Ritterman, Henry, Capt., 117-06 225th St., St. Albans.
Rochfeld, Morris, Capt., 1363 W. 6th St., Brooklyn.
Rock, Julius, Major, 27 Stone Rd., Rochester.
Rose, Oscar A., Capt., 235 E. 46th St., New York.
Rose, Richard A., Major, 15 Stephenson Blvd., New Rochelle.
Rosenberg, Jonas S., Capt., 300 W. 109th St., New York.
Rosenberg, Morris, Capt., 1929 Davidson Ave., Bronx.
Rosenthal, Quintin, Capt., Brook Lane, Peekskill.
Rosner, Samuel, 1st Lt., 2102 Bronx Park E., New York.
Rothenberg, Joseph G., Capt., 302 S. 1st Ave., Mount Vernon.
Rothstein, Isadore, Major, 1495 Popham Ave., Bronx.
Rubin, Herman, Lt. Col., 277 Eastern Parkway, Brooklyn.
Sabini, Cecil F., Capt., 603 Jefferson St., Hoboken.
Saunders, William, Capt., 385 2d St., Troy.
Sawyer, Charles D., Capt., 625 3d St., Brooklyn.
Schatken, Robert V., Capt., 233 E. 50th St., New York.
Schintzius, William C., Major, Boonville.
Schneider, Louis, Capt., 1515 Grand Concourse, New York.
Schwartz, Morris, Capt., 8 Cliff St., Beacon.
Schwartz, Samuel, Capt., 1925 University Ave., Bronx.
Schweitzer, Martin, Capt., 991 Freeman St., New York.
Scibetta, Charles T., Capt., 92 Fargo St., Buffalo.
Scroggin, Frederick R., Capt., Polyclinic Hosp., New York.
Seapy, Lewis F., Capt., 2041 Eucalyptus Ave., Long Beach.
Seelinger, George F., Capt., 611 Concord Ave., Williston Park.
Segal, Lester, Major, 239 Central Park West, New York.
Seidman, Herman H., Major, 760 Hunts Point Ave., Bronx.
Selkin, William, Capt., 2060 Mapes Ave., Bronx.
Sharkey, William F., Capt., 17-44 149th St., Whitestone.
Shapiro, Samuel D., Capt., 1295 Grand Concourse, Bronx.
Single, Charles M., Capt., 82 E. Main St., Wolcott.
Soehner, Bernard P., Major, 444 Magee Ave., Rochester.
Spencer, Gordon A., Major, 54 Decker St., Buffalo.
Spencer, Ralph F., Major, 555 Union St., Hudson.
Spinelli, Vincent A., Major, 43-39 39th Pl., Sunnyside, L. I.
Spitz, Leon J., Capt., 237 E. 20th St., New York.
Spitzer, Jos. M., Lt. Col., 109-05 72d Ave., Forest Hills.
Spitzer, Norman, Capt., 56 Hawthorne Ave., Yonkers.
Spring, David, Capt., 14-22 122d, College Point.
Steele, Frank E., Capt., 205 W. 138th St., New York.
Stevens, Eldred J., Capt., 51 Main St., Hammondsport.
Stevens, Ralph P., Capt., Oak Hill, Greene County.
Steyaart, Charles L., Lt. Col., 101 Broad St., Lyons.
Stork, Gerard J., Capt., 104-17 220th St., Queens Village, L. I.
Terry, Richard N., Capt., 25 Indian Church Rd., Buffalo.
Tichenor, Clifford J., Major, Crouse Irving Hosp., Syracuse.
Totero, Nestor J., Capt., 635 E. 211th St., Bronx.
Turano, William M., Major, 321 E. 200th St., Bronx.
Walzer, Eugene H., Capt., 99 Clove Rd., New Rochelle.
Weiner, Max, Capt., 214-02 Murdock Ave., Queens Vil.
Wirth, Herman E., Major, 43-02 21st Ave., Astoria, L. I.
Zuckerman, Herman C., Capt., 1714 Corona Park, Bronx.

North Carolina

Augustine, R. W., Capt., % N. C. Orthopedic Hosp., Gastonia.
Brown, Ivan W. Jr., Capt., Duke Hosp., Durham.
Cutchin, Joseph H. Jr., Capt., Box 202, Whitakers.
Fales, Robert M., Capt., 311 S. 5th St., Wilmington.
Helsabeck, Belmont A., Capt., Nissen Bldg., Winston-Salem.
Kennedy, Leon T., Major, 827 Missen Bldg., Winston-Salem.
Lebauer, Maurice L., Major, 1509 Madison Ave., Greensboro.
Robertson, Lloyd H., Capt., Country Club, Salisbury.
Salle, George F., Capt., Vanceboro.
Sox, Carl C., Lt. Col., Kenly.
Warwick, Hight C., Capt., 2320 Kirkpatruck Pl., Greensboro.
Winstead, Maurice B., Major, 312 Harvey St., Washington.

PHYSICIANS SEPARATED FROM SERVICE

North Dakota

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Radl, Robert B., Lt. Col., 301 Avenue C West, Bismarck.

Ohio

Ameter, Russell K., 1st Lt., 509 Oakwood, Bryan.
Andrus, Willard B., Capt., 2454 Kinsington Rd., Columbus.
Bauhof, Ned F., Capt., 1838 E. 101st St., Cleveland.
Berschalsky, Solomon, Capt., 410 Catherine St., Cincinnati.
Blaugrund, Charles, Capt., 4775 E. 86th St., Cleveland.
Bogue, Charles A., Capt., 276 Monroe St. N.W., Warren.
Bookwalter, Henry L., Capt., 35 N. Main St., Columbiana.
Brett, John D., Major, 10465 Carnegie Ave., Cleveland.
Bronson, Lewis H. Jr., Capt., Lakeside Hosp., Cleveland.
Brown, Benjamin S., Capt., Indian Creek Farm, Canfield.
Butner, C. O., Capt., Box A, Shiloh.
Caudill, Darrell C., Capt., 400 Moore St., Middletown.
Chappel, Merwin R., Major, Cor. Ohio & Watt Sts., Athens.
Clippinger, Conrad K., Capt., 1602 Grand Ave., Dayton.
Cocrel, William A., Capt., 809 Oak St., Wyoming.
Coddington, Oscar L., Capt., 978 Jaeger St., Columbus.
Daus, Homer J., Major, 6713 Superior Ave., Cleveland.
Del Vecchio, James J., Major, Rayland.
Donald, John H., 1st Lt., R. R. 2, Ripley.
Dornheggen, John H., Capt., 2317 Grandview Ave., Cincinnati.
Dowell, Lloyd L., Capt., P. O. Box, 442, Navarre.
East, William J., Capt., 2717 Fulton St., Apt. 4, Toledo.
Fberle, William H., Capt., 4500 Park Ave., Ashtabula.
Epler, Deane C., Capt., 295 W. Hubbard Ave., Columbus.
Felstkorn, Karl H., Capt., 286 S. Main St., Marion.
Fitzgerald, George P. Jr., Capt., 1317 Garfield Ave., Springfield.
Folger, Glen K., Lt. Col., 2179 Delaware Dr., Cleveland.
Folin, John J., Capt., 249 E. Robinson Ave., Barberton.
Fowler, Evan C., Major, 437 N. 18th St., Sebring.
Garcia, Charles T., Capt., 3328 Euclid Ave., Cleveland.
Good, Charles F., Major, 11333 Hessler Rd., Cleveland.
Grogan, Francis R., Major, 126 Scioto St., Urbana.
Gunn, Herman M., Lt. Col., 104 E. Walnut St., Ashland.
Hagen, John S. Jr., Major, 595 Howell Ave., Cincinnati.
Hanson, Kenneth B., Major, 2000 Columbia Pkwy., Cincinnati.
Hattenbach, Joseph F., Capt., 1308 Elmwood Rd., Rocky River.
Hauss, John F., Capt., 623 N. Main St., Celina.
Hengen, Henry E., Capt., 559 Hazel St., Amherst.
Hoffman, Charles R., Capt., 899 Lexington Ave., Cincinnati.
Horton, Robert J. M., Capt., 3215 Oak Rd., Cleveland Heights.
Jacobs, Herbert M., Lt. Col., 3034 Walton Ave., Cleveland.
Johnson, James B., Capt., 1350 W. 6th Ave., Columbus.
Keller, Bayard M., Capt., 1922 2d St., Cuyahoga Falls.
Kelly, Fred R., Lt. Col., 2010 E. 102d St., Cleveland.
King, Thomas A., Capt., 1610 Brainard Ave., Cleveland.
Klopper, Edward G., Capt., The Cleveland Clinic, Cleveland.
Light, Theodore L., Capt., 2670 Salem Ave., Dayton.
Loeb, William J., Major, 12805 Cedar Rd., Cleveland Heights.
Lotzoff, Harold A., Capt., 211 S. Woodlawn St., Lima.
Lulenski, Chester R., Capt., 1862 E. 101st St., Cleveland.
McWilliam, John M., Capt., 2846 Inwood Dr., Toledo.
Maglio, Gerard C., Capt., 44 Barker Ave., White Plains.
Meckstroth, Paul G., Major, New Knoxville.
Mills, James F., Capt., 6341 Beechmont Ave., Cincinnati.
Mowry, John H., Capt., 401 Jackson St., Conneaut.
Myers, Robert D., Capt., 915 Welton Ave., Columbus.
Neff, Garnet E., Capt., 3033 N. Hill Rd., Portsmouth.
Pinnell, Earl E., Capt., 21 S. Pleasant Ave., Osborn.
Rian, Carlos D., Capt., 738 Rotch Ave. N.E., Massillon.
Root, Harold E., Capt., 214 Southwest St., Bellevue.
Sebastian, Charles A., Capt., 2085 Harrison Ave., Cincinnati.
Singer, Merle K., Capt., 771½ N. Main St., Akron.
Suttle, Robert C., Capt., 1709 Grand Ave., Toledo.
Wertheimer, Lee C., Capt., 515 Maxwell Ave., Cincinnati.
Wiedemer, Hall S., 1st Lt., 20 E. 11th Ave., Columbus.
Wirtschafter, Z. T., Lt. Col., 3460 Menlo Rd., Shaker Heights.

Oklahoma

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Mills, Charles K., Lt. Col., 602 E. Creek St., McAlester.

Oregon

Haslinger, Joseph F., Major, 2365 N.W. Irving St., Portland.
Pease, Bradford N., Major, 2481 S.W. Sherwood Dr., Portland.
Ray, Leon F., Capt., 3426 N.E. 19th Ave., Portland.
Robb, Marvin J., Capt., 151 S. 6th St., Hillsboro.
Scales, Kenneth J., Capt., 3311 S.W. 10th St., Portland.
Skelley, Lee C., Major, 2226 N.W. Hoyt St., Portland.
Warrington, William R., Major, Univ. of Oregon, Portland.

Pennsylvania

Allen, Robert E., Col., 38 W. 3d St., Mount Carmel.
Axelrod, Bernard M., Capt., 1700 N. 57th St., Philadelphia.
Bailey, Frank R., Lt. Col., 209 Tennyson Ave., Pittsburgh.
Bealor, John A., Capt., % Hershey Hospital, Hershey.
Bello, Carmen T., Capt., 2036 S. 21st St., Philadelphia.
Biddle, John E., Capt., 212 Main St., Watsontown.
Biggins, James A., Capt., 420 N. Walnut St., Sharpsville.
Biskup, George E., Capt., 730 Main St., Mount Pleasant.
Blumberg, L. D., Lt. Col., Drake Hotel, 1512 Spruce, Philadel.
Boyer, George S., Capt., 318 W. Packer Ave., Bethlehem.
Braden, Frank R. Jr., Major, 1616 State Ave., Coraopolis.
Bretherick, Bernard S., Capt., Roulette.
Brohm, Charles G., Capt., Hawthorne.
Brown, Herman, Capt., 2201 N. 33d St., Philadelphia.
Browne, William C., Capt., Kinzua.
Bush, Leonard F., Lt. Col., Danville.
Buttermore, R. M., Major, 37 N. Bryant, Bellevue, Pittsburgh.
Cathcart, Hugh, Capt., Presbyterian Hosp., Philadelphia.
Ceraso, Louis C., Capt., 1721 5th Ave., Arnold.
Christman, Albert H., Major, 8 E. 5th St., Emporium.
Cohen, Merton E., Capt., 1314 Northhampton St., Easton.
Cooper, Franklin B., Lt. Col., 701 Allegheny Ave., Oakmont.
Cottom, Delmar C., Capt., 3403 Quay St., McKeesport.
Cowan, Thomas H., Major, 328 S. Camac St., Philadelphia.
Cramer, Earl D., Major, R. D. 2, Latrobe.
Di Stefano, Grimaldo C., Capt., 1302 Porter St., Philadelphia.
Donaldson, J. S., Major, Hawthorne Rd., Fox Chapel Borough, Pittsburgh.
Dutlinger, Robert P., 1st Lt., 18 Pennsylvania Ave., Mt. Union.
Echenberg, Max, Capt., 809 Chestnut St., Lebanon.
Etter, Lewis E., Major, Warrendale.
Evans, Harold H., Major, 402 Chestnut St., Berwick.
Everhart, Wilson C., Capt., Children's Hosp., Pittsburgh.
Fegley, Nathan A., Capt., 311 E. Main St., Schuylkill, Haven.
Fitts, William T. Jr., Capt., Univ. Pa. Hosp., Philadelphia.
Geyer, Harry M. Jr., Major, 166 Longue Vue Dr., Mt. Lebanon.
Gladney, James F. Jr., Major, Homer.
Gleason, Philip M., Major, 4111 Walnut St., Philadelphia.
Glinsky, George C., Major, Pa. State San., Hamburg.
Harris, Maurice N., Capt., 133 Carlisle St., Hanover.
Harrison, Milton, Major, 5118 N. Broad St., Philadelphia.
Heid, George J. Jr., Capt., Reading Hosp., Reading.
Helwig, Frederick G., Capt., 102 S. Madison St., Allentown.
Herbert, Michael J., Capt., 449 Sunbury St., Minersville.
Hollingsworth, John M., Capt., 23 Myrtle St., Girard.
Hutchison, Henry E., Major, 1101 Locust St., McKeesport.
Impink, Robert R., Lt. Col., Shillington.
Ingham, Albert J., Major, 315 W. Main St., Titusville.
Janjigian, Edward R., Major, Danville State Hosp., Danville.
Johnson, Benjamin H. Jr., Major, Robert Packer Hosp., Sayre.
Kabakjian, Ramond, Capt., 82 N. Lansdowne Ave., Lansdowne.
Kannapel, Allen R., Capt., 717 N. 1st St., Lehighton.
Kasser, Max D., Capt., 2800 W. Master St., Philadelphia.
Katz, Samuel H., Capt., 5811 Chester Ave., Philadelphia.
Krohn, Harold A., Major, 1330 Oak St., Lebanon.
Lang, H. B., Capt., 403 Pasadena Dr., Fox Chapel, Pittsburgh.
Laughlin, Robert M., Major, 513 Lorenz Ave., Pittsburgh.
Lavin, Frank C., Capt., 155 S. Main Ave., Scranton.
Lawlor, John M., Capt., 2314 W. Harold St., Philadelphia.
Leibold, Robert W., Capt., 225 Waldorf St., Pittsburgh.
Leinbach, Harvey D. Jr., Capt., 1101 Evergreen Rd., Reading.
McAndrew, Paul C., Major, 1628 Sanderson Ave., Scranton.
McCartney, George A., Capt., Box A, Seward.
McHugh, Joseph W. Jr., Major, 145 Fayette St., Johnstown.
McMahan, Joseph N., Major, 19 Laurel Ave., Washington.
McNabb, James R., Capt., Burnham.
McNeal, Samuel W., Capt., 741 Chestnut St., Columbia.
Malcolm, Donald C., Capt., Alexandria.
Maylath, Florian R., Capt., 508 E. 4th St., Bethlehem.
Maunz, Daniel H., Lt. Col., Thomas Rd., Bradford.
Mercer, Theodore L., Capt., 214 Pennsylvania Ave., Avondale.
Miller, Elmer E., Capt., Hellam.
Miller, Ira C., Col., 2711 Logan St., Camp Hills.
Mitrani, Jacques H., Major, 229 W. Front St., Berwick.
Mitterling, Robert C., Capt., 5731 Baltimore Ave., Philadelphia.
Myers, William C., Capt., 1239 Monterey St. N.W., Pittsburgh.
Novak, John G., Major, 112 Stratford Ave., Pittsburgh.
Pitchford, W. N., Major, 2736 Espy Ave., Dormont, Pittsburgh.
Porter, Edgar L., Major, Chester City Hosp., Embreeville.
Prescott, W. D., Capt., 193 S. Tulpehocken St., Pine Grove.
Rhoads, John P., Capt., 145 Baltimore St., Gettysburg.
Rizika, Stuart D., Capt., 863 S. George St., York.
Robertson, Harold F., Lt. Col., 246 Rockglen Rd., Penn Wynne.
Rubin, Jerome J., Capt., 1332 Devereaux Ave., Philadelphia.

PHYSICIANS SEPARATED FROM SERVICE

Pennsylvania—Continued

Sandhaus, Julius L., Major, 426 W. King St., Lancaster.
Sarraf, George J., Capt., 3701 Penn Ave., Pittsburgh.
Saul, Theodore J., 1st Lt., Centre St., Dushere.
Schultz, Edward J., Major, Claysburg.
Simpson, Robert C., Lt. Col., Box 325, Ridgway.
Sowers, John W., Capt., Fayetteville.
Staskiel, Louis J. Jr., Capt., 53 Coal St., Glen Lyon.
Steele, Frederick H., Major, Taylor Highlands, Huntingdon.
Steele, Ralph E., Capt., RFD 1, Carlisle.
Stevenson, Alfred S., Major, Oakdale.
Stoner, Robert R. Jr., Lt. Col., York Springs.
Strini, Joseph F., Capt., 1101 Church Ave., McKees Rocks.
Stutzman, C. M. Jr., Capt., 308 E. Central, S. Williamsport.
Swingle, James W., Capt., RFD 1, Box 186-A, Lake Ariel.
Taksa, David S., Capt., 308 2d St., Elizabeth.
Tobias, Carl A., Capt., 509 Main St., Forest City.
Turnblacer, Charles B., Major, 317 W. Penna St., Butler.
Uhde, George I., Major, Longwood Gardens, Kennett Square.
Vesely, John A., Capt., RFD 1, Fayette City.
Volkwein, Frederick W. W., Capt., 532 Station St., Bridgeville.
Williams, Donald D., Capt., 551 W. 26th St., Erie.
Williams, Herman J., Capt., 439 N. 11th St., Reading.
Wymer, Ralph M., Capt., Mercy Hosp., Pittsburgh.

Rhode Island

Clagett, Augustus H. Jr., Lt. Col., Rhode Is. Hosp., Providence.
Hoffman, Arthur O., Capt., 380 Lloyd Ave., Providence.
Maynard, Jean M., Capt., 42 Curson St., West Warwick.

South Carolina

Allgood, James E., Capt., Inman.
Brooks, Thomas G., Lt. Col., Highland Park Dr., Aiken.
Lacey, William H., Capt., City Hall, Georgetown.
Plotkin, Oscar M., Major, 9 Cedar Terrace, RFD 4, Columbia.
Sweatman, Carl A., Capt., 137 Harden St., Columbia.

Tennessee

Chamberlain, Douglas, Capt., 811 W. Brow Rd., Lookout Mt.
Redmon, John J., Capt., John Gaston Hosp, Memphis.
Stem, William A., Capt., 3605 12th Ave., Chattanooga.

Texas

Anderson, James P., Capt., 1403 Pine St., Brady.
Cohen, Raymond, Major, 2204 Bellefontaine, Houston.
Cuen, Mario J., 1st Lt., 1122 Montana St., El Paso.
French, Cecil M., Capt., Methodist Hosp., Dallas.
Garrett, L. M., Lt. Col., 612 Med. Prof. Bldg., Corpus Christi.
Gillespie, Charles H., Capt., Scott & White Clinic, Temple.
Griffin, Frank S. Jr., Major, Liberty.
Guinn, James E., Capt., 1063 E. Terrell Ave., Fort Worth.
Hawker, LaVerne J., Major, 195 Alston, Fort Worth.
Heger, Frank F., Major, Shiner.
Kaufman, Herschel J., Capt., 3618 5th St., Port Arthur.
Malakoff, Morris E., Capt., O.O. Box 552, Laredo.
Moser, Emil R., Major, 109 N. Main St., Gladewater.
Pierson, Marcus A., Major, 1009 N. Galveston.
Rowe, Robert J. Jr., Capt., Kaufman.
Scales, John G., Capt., 328 W. Colorado St., Dallas.
Shacklett, Ernest D. Jr., Capt., 808 E. Quincy St., San Antonio.
Shapiro, Isadore A., Major, 17 G Inf. Post, Fort Sam Houston.
Still, Oscar W., Major, 6621 Lakewood Blvd., Dallas.
Swanson, Wayland R., Lt. Col., P. O. Box 66, Taylor.
Ward, Irvan M., Capt., 1612 Park St., Greenville.

Utah

Muir, Everett B., Major, 1408 Federal Way, Salt Lake City.

Virginia

Allen, Herbert C. Jr., Capt., 4015 Park Ave., Richmond.
Brantly, Edgar C. Jr., Capt., 124 Lady Astor St., Danville.
Burke, James O., Major, 204 E. Franklin St., Richmond.
Casscells, Samuel W., 1st Lt., 34th St., Norfolk.
Damron, Harold B., Lt. Col., 508 Park St., Bristol.
Davis, Charles F. Jr., Lt. Col., 2831 Melrose Ave., Roanoke.
Dunn, Edward T. Jr., Capt., 310 Ridgeway St., Clifton Forge.
Harkrader, Charles J. Jr., Capt., 115 Solar St., Bristol.
Marsella, John J., Capt., 1200 E. Marshall St., Richmond.
Paine, Wilmer H., Major, 112 3d St. N.W., Charlottesville.
Pinckney, Morton M., Lt. Col., 3104 Monument Ave., Richmond.
Rudolph, Robert R., Capt., 328 Avenham Ave., Roanoke.
Staley, Joseph S., Major, Marion.
Stone, Carey A. Jr., Capt., 411 E. Maryland Ave., Crewe.
Tyler, Gilmar R., Major, Med. Coll. of Va., Richmond.
Zack, Frank A., Capt., 328 56th St., Newport News.

Washington

Altman, Milton E., Capt., Box 108, Sedro Wooley.
Angland, Thomas A., Capt., 805 W. Yakima Ave., Yakima.
Bledsoe, Clarence W., Major, 522 McGilvra Blvd., Seattle.
Brakel, Karl A., Major, 4734 Thackeray Pl., Seattle.
Burns, John K., Major, 8 N. Howard St., Spokane.
Cash, George C., Lt. Col., Capital Apt., Olympia.
Coleman, Warren R., Capt., 9424 E. E St., Tacoma.
Compton, David W., 1st Lt., Vashon, Kind Co.
Dorland, Edison G., Major, 7710 Ravenna Ave., Seattle.
Drewelow, Kenneth R., Capt., Cort Rd., Kirkland.
Flynn, John E., Major, 1617 Rucker Ave., Everett.
Forman, Burnett B., Major, Govey Bldg., Shelton.
Harriage, Robert R., Major, 224 Spruce Ave., Montesano.
Hartzell, Homer V. Jr., Lt. Col., 3935 Sunnyside Ave., Seattle.
Hines, Harold C., Capt., 716 W. Yakima St., Pasco.
Larson, Charles P., Lt. Col., Tacoma GH, Tacoma.
Lynch, Edward D., Major, 1212 W. Yakima Ave., Yakima.
McDowell, John W., Major, 7753 33d Ave. N.W., Seattle.
Schoolnik, Max L., Capt., 550 Stimson Bldg., Seattle.
Schroeder, Herman J., Major, 1111 Boren Ave., Seattle.
Thompson, Thomas D., Major, 706 E. Highland, Spokane.
Way, John D., 1st Lt., 731 Alder, Seattle.
Wood, Quentin Lee, Capt., 2512 Roanoke, Seattle.

Wisconsin

Bell, Robert S., Major, St. Mary's Hosp., Madison.
Birge, Edward A., Lt. Col., 2554 Kendall Ave., Madison.
Black, Samuel B., Capt., 7331 Wellauer Dr., Wauwatosa.
Bunts, Robert C., Major, Wisconsin Gen. Hosp., Madison.
Conen, Warren J., Major, 908 N. 12th St., Milwaukee.
Feiman, Lawrence H., Capt., 2518 N. Stowell Ave., Milwaukee.
Flannery, John V., Capt., 204 Franklin St., Wausau.
Hartman, Alexander S., Capt., 622 W. Mitchell St., Milwaukee.
Hitz, John B., Lt. Col., 2908 N. Stowell Ave., Milwaukee.
Karberg, Richard J., Capt., 2108 Rowley Ave., Madison.
Konz, Stephen A., Capt., 50 Bellaire Ct., Appleton.
Leininger, Alfred T., Capt., Milwaukee.
Milchen, Carl, Capt., Prairie du Chien.
Nowack, Lewis W., Lt. Col., Watertown.
Poindexter, Marlin H. Jr., Capt., Mil. Child. Hosp., Milwaukee.
Olsen, Leonard C. J., Capt., Box 25, Delafield.
Qualls, Charles L., Capt., 146 Oak Grove St., Juneau.
Rechlitz, Ervin T., Lt. Col., Milltown.
Sand, Harry H., Capt., 2453 N. 17th St., Milwaukee.
Schlomovitz, Harry H., Major, 3d & LaSalle St., Barron.
Schneider, Charles S., Capt., 2831 N. 51st St., Milwaukee.
Simenson, Raymond S., Capt., Valders.
Snodgrass, Herbert M., Capt., 716 W. Dayton St., Madison.
Solberg, Marvin E., Capt., 631 N. Church St., Richland Center.
Vedder, Charles A., Major, 716 Highland Ave., Marshfield.
Vetter, Edward W., Capt., Brandon.
Watry, Theodore D., Capt., 1950 W. Nash, Milwaukee.
Weisfeldt, Simon C., Capt., 3036 N. 62d St., Milwaukee.
Wilets, Jack B., Capt., 3488 N. Murray Ave., Milwaukee.
Winkler, Raymond J., Lt. Col., Hilbert.
Winsauer, Henry J., Capt., 229 Orchard Rd., Kohler.

Wyoming

Giovale, Silvio J., Major, Rock Spring.

Hawaii

Chang, Hon C., Capt., Lihue, Kauai.
Chunming, Archie, Major, 164 S. Vineyard St., Honolulu.
Depp, Donald S., Capt., Waipahu Hosp., Waipahu, Oahu.
Kainuma, Richard T., Capt., Waialua, Oahu.
Kawasaki, Isaac A., Major, 307 N. Kukui St., Honolulu.
Luke, Leslie N. Y., Capt., 1189 Bethel St., Honolulu.
Stilson, Homer O., Major, Waianae.
Stitt, Albert, Capt.
Tong, Fook H., Major, 1927 Coyne Ave., Honolulu.

Puerto Rico

Jimenez, Roberto J., Major, 97 Ponce de Leon Ave., Santurce.
Matta, Enrique L. J., Capt., Fajardo.
Nieves-Colon Juan de la Cruz, Capt., 33 General Contreras St., Cayey.
Oms, Luis R., Major, Municipal Hosp., Humacao.

CHANGES

Cauble, Wilbur G., Capt., Benedict.
Girod, Charles I., 1st Lt., Cambridge.
Rosenthal, Arthur D., Capt., Vt. Adm. Facility, Lyons.

Foregoing officers have not been relieved from active duty.

MEDICAL OFFICERS RELEASED BY THE NAVY

The figures in the Personnel Division of the Bureau of Medicine and Surgery indicate that 1,259 medical officers have been released and their records closed in this bureau, and information from the Bureau of Naval Personnel indicates that 1,635 medical officers are under orders for release from active duty, making a total of 2,894 separated or under orders for separation as of Dec. 1, 1945. The critical score effective Jan. 1, 1946 makes 5,583 medical officers eligible for separation. This number does not include medical officers released by reason of hardship, limited shore duty or affiliation with teaching institutions and hospitals.

NAVAL RESERVE MEDICAL OFFICERS RECOMMENDED FOR RELEASE FROM ACTIVE DUTY

Alabama

Callaway, Raymond R.....Birmingham
Gaillard, Samuel S.....Mobile
Howard, John M.....Autaugaville
Littlejohn, Wilmot S.....Birmingham
Meeker, William R.....Mobile
Parsons, William C.....Birmingham
Rumpanos, Socrates N.....Mobile
Smith, Gordon R.....Ozark
Thompson, Clyde T.....Wilson Dam

Arizona

Piepergerdes, Clarence C.....Bisbee

Arkansas

Barrett, Edward R.....Jonesboro
Calcote, Royal J.....Little Rock
Krock, Frederick H.....Ft. Smith
Stigler, Stephen L.....Little Rock
Stone, Wayne B.....Waldenburg

California

Adams, Robert M.....San Diego
Albertson, Henry K.....San Diego
Brown, Dewey F.....Redwood City
Burke, George W.....San Francisco
Cardwell, John L..Piedmont (Oakland)
Chambers, Jack V.....Sacramento
Costolow, William E.....Los Angeles
Davis, John P.....Santa Ana
Ellmore, Lewis F.....Los Angeles
Emerson, Charles V. A.....Los Angeles
Ewing, John P.....Los Angeles
Gilman, Philip K. Sr.....San Francisco
Goodcell, Ross A.....Los Angeles
Green, Marion M.....Stockton
Holman, Emile F.....San Francisco
Holmes, Olin M.....San Mateo
Johnson, Clark M.....San Francisco
Jones, George W.....Pomona
Kaplan, Harry E.....Lon Angeles
Lauer, Calvin A.....Long Beach
Leef, Edward E.....San Francisco
Lees, Floyd E.....Hanford
McDowell, Arthur J.....San Francisco
McElhinney, Philip P. B.....Bellflower
Mansfield, Raymond J.....San Francisco
Markthaler, Edward L.....Santa Barbara
Marshall, James M.....San Marino
Parker, Leon O.....San Francisco
Patterson, John K.....Santa Barbara
Pendergrass, Clayton I.....Clovis
Pentz, Clarence R.....Long Beach
Pressman, Joel J.....Los Angeles
Rapaport, Walter.....Ontario
Reeder, Charles W.....Long Beach
Reynolds, Lloyd R.....San Francisco
Rhind, Ralph M.....Hermosa Beach
Rogers, William L.....San Francisco
Rusche, Carl F.....Hollywood
Russell, William M.....San Diego
Sherman, Samuel R.....San Francisco
Simon, Julius.....Hollywood
Sokolow, Maurice .....San Francisco
Stanley, Leo L.....San Rafael
Steele, Edson H.....Los Angeles
Stewart, John E.....Alameda
Stofer, Dar Delos.....San Diego

Colorado

Barnard, Hamilton I.....Denver
Eckhardt, George C.....Boulder
Orr, Joseph K.....Craig
Thompson, John W.....Pueblo
Weeks, Paul R.....Denver

Connecticut

Baker, Clifford C.....Hartford
Baron, Shirley H.....New London
Bidgood, Charles Y.....Hartford
Brown, Norton S.....Newton
Crampton, Clair B.....Middletown
Crosby, Edward H.....Hartford
Eckelberry, Niel E.....Georgetown
Green, William F.....Newton
Kornblut, Alfred.....Bridgeport
Lampson, Rutledge S.....Hartford
Lewicki, Edward S.....Waterbury
Mayo, Elliott R.....Waterbury
Moran, James P.....New London
Nesbitt, Samuel.....New Haven
Noble, Philip C.....Missoula
Roberts, Frederick W.....Hamden
Stevens, Marvin A.....Harwinton
Ward, Robert H.....Darien
Wilson, George C.....Wallingford

Delaware

Farr, Lee E.....Wilmington
Martin, John P.....Camden
Squires, Millard F. Jr..Richardson Park
Thomison, Jamuel J. Jr.....Wilmington

District of Columbia

Cohen, Roger S.....Washington
Grove, Pembroke T.....Washington
Hopkins, Gerald A.....Washington
Kellerman, Edgar A. P....Washington
Kreuzburg, Harvey F.....Washington

Florida

Andrews, Edwin H.....Gainesville
Bedell, Sullivan G.....Jacksonville
Bradley, James A.....Petersburgh
Cline, Abe.....Jacksonville
Gemmill, Chalmers L.....Pensacola
Hebard, Charles E.....St. Petersburg
Jack, Ralph W.....Miami Beach
Kauders, Ferdinand H.....Miami Beach
Martin, Douglas D.....Tampa
Reese, Homer A.....Miami
Richards, Ferdinand.....Jacksonville
Riley, Julian G.....Jacksonville
Rudolph, Councill C.....Petersburg
Swift, Edwin C.....Jacksonville
Vallotton, Joseph R.....Dayton Beach
Weiland, Arthur H.....Coral Gables
Williams, William L.....Pensacola
Wright, Claude B.....St. Petersburgh

Idaho

Mack, Quentin W.....Boise

Illinois

Abrams, Jack S.....Chicago
Drennan, George L.....Jacksonville
Holm, Alf J.....Chicago
Imbiorski, Stanley J.....Chicago
Westenberger, Lorenz H.....Chicago
Zieman, Stephen A.....Chicago

Indiana

Baxter, Neal E.....Bloomington
Beard, Paul H.....Indianapolis
Cohen, Bernard W.....Indianapolis
Duemling, Arnold H.....Fort Wayne
Rhamy, Arthur P.....Wabash
Rust, Byron K.....Indianapolis
Scott, Harry V.....Fort Wayne
Slominski, Harry H.....South Bend
Waite, Richard R.....Lafayette
Yegerlehner, Roscoe S.....Kentland

Iowa

Ady, Albert E.....Marengo
Anderson, Stanley N.....Onawa
Bunch, Harold M.....Shenandoah
Campbell, Walter V.....Oskaloosa
Edwards, Sydney K.....Red Oak
Fullgrabe, Emil Albert.....Indianola
George Everett M.....Des Moines
Gilfillan, George W.....Bloomfield
James, Audra D.....Des Moines
Jongewaard, Albert J.....Jefferson
Leinbach, Samuel P.....Belmond
Lohr, Phillips E.....Churchdan
McGilvra, Raymond I....Guthrie Center
McKitterick, John C.....Burlington
Paige, Ralph T.....La Porte City
Sorenson, Aral C.....Davenport
Werthmann, Paul A.....Anthon

Kansas

Ashley, Byron J.....Topeka
Byers, Philip L.....Kansas City
Caldwell, George M.....Manhattan
Parmley, Charles C.....Hutchinson
Porter, John M.....Concordia
Royer, Charles A.....Coffeyville
Royse, George T.....Coffeyville
Speer, Frederic A.....Kansas City
Speer, Leland N.....Kansas City

Kentucky

Adair, Walter M.....Russellville
Akin, William E. Jr.....Paintsville
Bowen, Joseph A.....Louisville
Fuller, James T.....Mayfield
Goldberg, Harry.....Matthews
Keller, William K.....Louisville
McClure, George M., Jr.....Danville
Overstreet, Samuel A.....Louisville

Maine

Apollonio, Howard L.....Camden
Cuneo, Kenneth J.....Kennebunk
Hanlon, Francis W.....Portland
Hinman, Havilah E.....Orono
Smith, Kenneth E.....Portland

Maryland

Allison, Robert H.....Baltimore
Bowers, John Z.....Baltimore
Broyles, Edwin N.....Baltimore
Goldsmith, Harry .....Baltimore
Goodman, Jerome E.....Baltimore
Hellman, Louis M.....Baltimore
O'Donovan, Charles .....Baltimore
Schlesinger, George G.....Baltimore
Williamson, Edgar P. II.....Catonsville
Wilson, Edward C. Jr.....Edgewater

MEDICAL OFFICERS RELEASED BY THE NAVY

NAVAL RESERVE MEDICAL OFFICERS RECOMMENDED FOR RELEASE FROM ACTIVE DUTY

Massachusetts

Abrams, Archie A.....Brookline
Belcher, Charles D., J.....Winthrop
Bigelow, Robert B.....Milton
Boguniecki, Stanley J.....Westfield
Carr, Frank B.....Worcester
Chamberlin, Harold A.....Newtonville
Cordray, David P.....Boston
Cozza, Lawrence F.....Sommerville
Duncombe, Alfred L.....Brockton
Dunphy, Edwin B.....Chestnut Hill
Farnsworth, Dana L.....Williamstown
Faulkner, James Morrison.....Brookline
Fay, John H.....Boston
Forbes, Wilfred W.....Gloucester
Fox, Samuel .....Holyoke
Harris, Herbert I.....Westport Point
Helgesson, Uno H.....Longmeadow
Hilton, Philip F.....Leominster
Jacobs, Lee D.....Mattapan
Johnson, Charles I.....Boston
Kitts, Albert W.....Boston
Kontoff, Henry A.....Newton Center
Kuchar, Benjamin E.....Brighton
Kunian, David .....Lynn
Lamphier, James A.....Newton
Levenson, Walter S.....Brighton
Miller, Julius Y.....Chelsea
Olson, Joseph M.....Westboro
Osterheld, Roger G.....Concord
Pratt, Theodore C.....Boston
Roberson, Tracey L.....Ware
Schirmer, Adelbert.....Roslindale,
Sullivan, Gerald J.....Worcester
Sullivan, William J.....Milton
Thornton, John J.....Boston
Volk, Ralph .....Brookline

Michigan

Adams, Frank A.....Grand Rapids
Blaine, Max.....Detroit
Campbell, Charles A.....Detroit
Dimitroff, Sim P.....Ann Arbor
Fandrich, Theodore S.....Detroit
Grimaldi, Gregory J.....Detroit
Hilt, Laurence M.....Grand Rapids
Hoogerhyde, Jack.....Grand Rapids
Johnson, Lester J.....Ann Arbor
Olsen, Richard E.....Pontiac
Ottaway, John P.....Grosse Pt. Farms
Pfeiffer, Carl C.....Detroit
Reagan, Robert E.....Benton Harbor
Rector, Edgar M.....Ann Arbor
Richter, Harry J.....Saginaw
Williams, Richard J.....Monroe
Wilson, Merton C.....Detroit

Minnesota

Bagley, Charles M.....Duluth
Becker, Sidney F.....Minneapolis
Bird, Lee C.....Minneapolis
Borowicz, Leonard A.....Minneapolis
MaGath, Thomas B.....Rochester
Mayne, Roy M.....Duluth
Miller, Hugo E.....Minneapolis
Naslund, Ames W.....Minneapolis
Nielsen, Alvin M.....Northfield
Peterson, John H.....Duluth
Rosenholtz, Burton.....St. Paul
Rudie, Peter S.....Duluth
Schulze, William M.....Minneapolis
Shimonek, Stewart W.....St. Paul
Steinberg, Charles L.....St. Paul
Sweetster, Horatio B. Jr.....Minneapolis
Sybilrud, Hjalmer W.....Bricelyn
Tibbetts, Mark H.....Duluth
Vogel, Howard A. L.....New Ulm
Walter, Clarence W.....St. Paul
Wheeler, Daniel W.....Duluth

Mississippi

Coffey, John D. Jr.....Vicksburg
Fiegel, Samuel A.....Sturgis
Howard, Homer L.....Winona
Jones, Isaac H.....Meridian
Lynch, Robert H. F.....Marrs
Stallworth, William L.....Columbus
Whittington, Homer A.....Natchez

Montana

Bennett, Arthur A.....Roundup
Delaney, James R.....Kalispell
Hawkins, Thomas L.....Helena
Key, Roy Whitfield.....Missoula
Noble, Philip C.....Missoula
Shaw, John A.....Billings

Nebraska

Clarke, Harvey L. Jr.....North Platte
Glazier, McCleery.....Broken Bow
Iwersen, Frank J.....Omaha
McLaughlin, Charles W., Jr.....Omaha
Royer, Howard.....Grand Island
Underwood, George R.....Lincoln
Waggoner, Charles M.....Omaha

New Hampshire

Hand, Francis J.....Epping
Hunter, Ralph W.....Hanover
McCarthy, Joseph M.....Concord

New Mexico

Johnson, Lionel W.....Roswell
Lundgren, Rupert W.....Las Cruces
Poppen, Mayo J.....Raton
Wilson, Robert B.....Albuquerque

New York

Acken, Henry S. Jr.....Brooklyn
Anopol, George .....Yonkers
Blaisdell, Harold A.....Jamestown
Blum, Lester.....New York
Boggs, Robert.....New York
Callahan, Edward T.....New York
Carter, James D.....New York
Culver, Clair H.....Falconer
Curran, Frank J.....New York
Donehue, Francis M.....New York
Duryea, Garrett De Nyse.....Glen Cove
Ewing, James H.....New York
Fackler, Charles L.....New York
Gerstley, Manfred J.....Lynbrook
Giustra, Frank X.....Brooklyn
Harris, Harold J.....Essex County
Hazzard, Charles T.....New York
Jaeger, Gern W.....Niagara Falls
Jameson, Edwin M.....Saranac Lake
Keenan, Robert J. A.....Bronx
Kees, Walter T.....New York
Lauterbach, Chester H.....Rochester
La Vine, Joseph J.....Baldwin
Macrobert, Russell G.....Larchmont
McCabe, Edward J.....New York
Miner, Theodore R.....Brooklyn
Murphy, Wallace B.....New York
Nahon, Joseph R.....New York
Napp, Emol E.....New York
Noonan, James E.....Yonkers
Pelow, William E.....Syracuse
Pendleton, Raymond K.....Brooklyn
Short, James J.....Forest Hills, L. I.
Siegartel, Morris .....Brooklyn
Tripler, Samuel.....New York
Trombetta, Alessandro .....Buffalo
Twaddell, Donald N.....Scarsdale
Weinstock, Harry I.....New York
Wetzen, Delmo L.....Jamestown
Witkin, Leonard E.....Brooklyn
Yarington, Charles T.....Moravia

North Carolina

Atkins, Junius Jr.....Raleigh
Britt, Charles S.....Charlotte
Brown, Landis G.....Southport
Bunch, Charles.....Charlotte
Burwell, Walter B.....Henderson
Graham, Charles P.....Wilmington
McAneill, James H...North Wilkesboro
Myers, Richard T.....Winston-Salem
Norfleet, Ashley C.....Tarboro
Nowlin, George P.....Charlotte
Parrot, George F.....Kinston
Schoenheit, Edward W.....Asheville
Smith, Edward B.....Elizabeth City
Stein, Ephraim.....Brooklyn
Stewart, Daniel N., Jr.....Hickory
Weeks, Carnes.....New York

North Dakota

Hagan, Edward J.....Williston
McIntosh, Hugh A.....Minot

Ohio

Beall, Neil P.....Cleveland
Birbeck, Norman J.....Dayton
Jacoby, Ben E.....Newark
Keating, Robert A.....Columbus
Keheler, Michael F.....Springfield
Northup, Spence W.....Toledo
Pfeiffer, Charles L.....Cincinnati
Root, Joseph C.....Chagrin Falls
Sage, Harry M.....Columbus
Salter, Herbert W.....University Hts.
Tagett, Harry A.....Ashtabula
Warm, Herbert.....Hamilton
Weaver, Howard B.....Canton
Webster, Frank B.....Bellefontaine
Weintraub, Josef D.....Cincinnati
Weir, William C.....Cleveland Hts.
Wiggers, Lowe H., Jr.....Wyoming
Witwer, Russell G....Cleveland Heights
Wright, Orville M.....Dayton
Yarris, William F., Jr.....Fostoria
Zeve, Harman S.....Youngstown

Oklahoma

Buchan, William H.....Tulsa
Coker, Battey B.....Durant
Jacobs, Raymond G.....Enid
Little, Aaron C.....Minco
Stone, Samuel N. Jr....Oklahoma Way

Oregon

Johnson, Alstrup N.....Roseburg
Massey, George D.....Klamath Falls
Rankin, Frederick O.....Portland
Robinson, Edwin G.....Portland
Southard, Durward W.....Baker
Spangler, Paul E.....Portland
Steiner, Milton B.....Portland
Stratford, Eldridge W.....Portland

Pennsylvania

Bachman, Carl E.....Philadelphia
Buck, William A. A.....Philadelphia
Hatch, Joseph C.....Johnston
McGrail, Matthew A.....Bradford
Robbins, Frederick R.....Bryn Mawr
Stango, Victor O.....Philadelphia
Tompkins, Winslow T.....Philadelphia
Ulrich, Henry F.....Middleburg
Weniger, Frederick L.....Waymart
Willetts, Arthur T.....Verona

Tennessee

Andrews, Charles G.....Memphis
Moore, Moore, Jr.....Memphis
Tendler, Morton J.....Memphis
Williams, Marvin L.....Memphis

ORGANIZATION SECTION

Official Notes

CONFERENCE OF STATE SECRETARIES AND EDITORS

The Conference of State Secretaries and Editors, which was originally scheduled to be held in November 1945, will be held at the offices of the American Medical Association in Chicago on Friday and Saturday, Feb. 8 and 9, 1946. Two sessions of the conference will be held on Friday, February 8, one session at 10 a. m. and the other on the afternoon of that day. On the evening of February 8 a program intended to be of particular interest to the editors of state medical journals will be presented. The concluding session of the conference will be held at the offices of the American Medical Association on the morning of Saturday, February 9.

Officers of constituent state medical associations and of component or district medical societies will be heartily welcome to attend the conference. All who expect to attend are urged to make necessary arrangements for transportation and for hotel accommodations at the earliest possible time.

NEW APPOINTMENT

Dr. A. Ray Dawson, who was recently relieved from service with the rank of lieutenant colonel, has been appointed assistant to the Secretary of the Council on Industrial Health of the American Medical Association, effective November 15. Dr. Dawson will be in charge of workmen's compensation and rehabilitation. Prior to his association with the reconditioning and rehabilitation program of the Army Air Forces, Dr. Dawson was affiliated with the Jefferson Standard Life Insurance Company, Greensboro, N. C., and served as medical officer in the U. S. Civil Service Commission, Washington, D. C. He graduated at the Medical College of Virginia, Richmond, in 1929.

Washington Letter

(From a Special Correspondent)

Dec. 17, 1945.

Committee Named to Coordinate Government Medical Services

President Truman has named Dr. Harold W. Dodds, president of Princeton University, head of a committee to coordinate the government medical services and to find ways of increasing medical facilities of the Veterans Administration. Also Major Gen. Paul R. Hawley, acting Surgeon General of the Veterans Administration, has selected eleven doctors, headed by Brig. Gen. Elliott C. Cutler, to serve as chief consultants to the agency. The consultants will select leading doctors throughout the country to work with staff physicians on a part time basis. The move is planned to insure the highest medical standards for hospitalized veterans. General Cutler is Moseley professor of surgery at Harvard Medical School and is attached to the Peter Bent Brigham Hospital in Boston.

Medical Center Bill to Be Modified

House District Committee members have indicated informally that they will amend the District of Columbia hospital bill, passed by the Senate, so that the entire initial cost would be borne by the federal government. The Senate had asked that private hospitals affiliating with the center pay one third of the initial cost and repay the other two thirds over fifty years. Alternate drafts of the measure are being prepared, and Representative Dan McGehee, Democrat of Mississippi, committee chairman, hopes to get action on the bill soon. The proposed District medical center has been the subject of outspoken testimony. Senator Millard Tydings, Democrat of Maryland, has asked for consolidation of the Twelfth District private hospitals, calling them "antiquated, rat and roach ridden, uneconomical and dangerous fire traps." He estimated the cost of the proposal at approximately $12,000,000. Major Gen. Philip B. Fleming, Federal Security Agency administrator, also asked for support of the measure, stating that the government might wholly finance an $18,000,000 medical center on a 25 acre site to be selected. He said the center would include 1,500 beds, a nurses' home, a school and parking space for 500 cars. It would be supervised by a corporation formed by agreement between the government and private interests. General Fleming said that Washington now has a 1,700 bed hospital shortage.

Senate Approves Federal Hospital Aid Program

The Senate has voted its approval, and similar action is sought in the House, on the federal hospital aid program, which is expected to provide $700 million in new construction during the next five years. The committee rejected a series of amendments offered by Senators Murray, Democrat of Montana, and Wagner, Democrat of New York, which they claimed would make the bill conform with President Truman's recent congressional message on a national health program. Senators Hill, Democrat of Alabama, and Taft, Republican of Ohio, worked together to defeat the Murray-Wagner proposals, which they said would remove proper state control of the program.

Influenza Vaccine Used by Army Is on Sale to Civilians

The vaccine used to vaccinate all army personnel this fall and winter against influenza types A and B is now on the market for civilian use. The vaccine is reported to be effective against the type B virus now causing outbreaks throughout the country. A check with producers reveals that Eli Lilly & Co., the Lederle Laboratories and the Pitman-Moore Company have the vaccine available now for civilian use. Pitman-Moore states that all its branches are being supplied. Parke Davis will have the vaccine available to civilians before January. The Squibb vaccine will be available for civilian use about February 15.

Bradley Won't End Segregation in Veterans' Hospitals

Demands of Negro leaders that segregation of white and Negro veterans in some Southern hospitals be ended has been refused by Veterans Administrator Omar N. Bradley. General Hawley, acting Surgeon General, advised General Bradley that the Veterans Administration could not be used as an agency for social change and must follow local customs. A delegation of twenty-five Negro organizations sought an interview with General Bradley to present their case. A survey recently conducted under General Bradley's orders shows that of the ninety-seven Veterans Administration hospitals seventeen do not accept Negroes except in emergency and that twenty-four have separate wards for Negroes. One hospital, at Tuskegee, Ala., accepts only Negroes. The remaining fifty-five make no distinction.

Warning by Vannevar Bush that Nation Faces Deficit of Scientific Capital

A short-sighted draft policy nearly wrecked the scientific side of the American war effort, states Dr. Vannevar Bush in his annual report as president of the Carnegie Institution of Washington. He said we are still drafting scientists and students of science, even though the war is over. The United States, he declared, drew so heavily on its "scientific capital" during the war that it now faces a "serious deficit" in young scientists. Many young scientists were taken out of laboratories during the war, and "we also sacrificed the future to immediate needs more than did any of our allies or, indeed, our enemies, by halting our processes of advanced education." He declared the nation must educate for scientific research all the brilliant young minds it can find in order to keep abreast of other countries.

AMERICAN RED CROSS DISTRIBUTION OF SURPLUS DRIED BLOOD PLASMA

NOTE.—This plan for the distribution and use of the surplus plasma has been presented to and concurred in by the Association of State and Territorial Health Officers, the American Medical Association and the American Hospital Association.

G. Foard McGinnes, M.D., National Medical Director.

During the war, large quantities of blood were given by the American people through the American Red Cross from which dried plasma was prepared for the armed forces. The supply of this material was predicated on the needs of the Army and the Navy for a long and costly war. Because of an earlier cessation of hostilities than was reasonably to be expected in both the European and the Pacific theaters, there is now in the hands of the Army and the Navy a quantity of dried plasma which is in excess of their needs during the anticipated useful life of the plasma, namely five years from the date of processing. According to army and navy estimates, the available surplus amounts to approximately one and a quarter million packages at the present time.

The transfer to the American Red Cross of dried plasma declared surplus by the Army and the Navy is provided for by Public Law 457 of the 78th Congress, approved Oct. 3, 1944. The pertinent portion of this law, section 11 (f), reads as follows:

"No surplus property which was processed, produced or donated by the American Red Cross for any government agency shall be disposed of except after notice to and consultation with the American Red Cross. All or any portion of such property may be donated to the American Red Cross, upon its request, solely for charitable purposes."

Under the foregoing provision of Congress, a formal request was made to the Army and the Navy that all surplus plasma be transferred to the American Red Cross. This action was taken on the ground that the American Red Cross has a responsibility to the American people to assure that plasma and other derivatives of the blood voluntarily contributed for the members of the armed forces be utilized to the best advantage and not wasted or offered for sale or barter. In making this request, it was proposed that any surplus should be returned to the American people, who had made these supplies of plasma possible, for use in veterans' hospitals and in civilian medical practice. This proposal was accepted by the Army and the Navy, and accordingly they will transfer to the American Red Cross all available surplus stores of plasma and other blood derivatives.

In developing a plan for the distribution of this material it was felt that the first consideration was that the needs of the Veterans Administration be met. We therefore discussed the matter with General Hawley and arranged to provide him with the amount of plasma which he has estimated will be required for use in Veterans Administration facilities during the next five years.

In planning for distribution and use in civilian medical practice there were three factors which we believed to be of particular importance: The distribution should be carried on in such a manner as, first, to assist in making possible an accurate determination of the needs for blood and blood derivatives in the various parts of the country; second, to strengthen and stimulate the development of already established state and local civilian blood and blood derivatives programs, and, third, to demonstrate the value of such programs and thus stimulate their establishment where they do not already exist.

The surplus dried plasma amounts to about one and a quarter million packages, which it is estimated will meet civilian needs for approximately two years. We are arranging for warehouse storage space in each of our five areas, the headquarters of which are located at New York City, Alexandria, Va., Atlanta, Ga., St. Louis and San Francisco. The available plasma will be apportioned to each of these areas on a population basis and supplied to state departments of health for distribution without charge to all physicians licensed to practice medicine and surgery, and to all acceptable hospitals for use in the treatment of any patient without charge for the product. This plan is similar to that which the state departments of health have been following in distributing the surplus immune serum globulin made available by the American Red Cross for the prophylaxis and modification of measles.

The area medical directors of the American Red Cross will make arrangements with state departments of health for a satisfactory plan for the distribution of plasma within each state, in consultation with representatives of the state medical society and state hospital association. The American Red Cross will then make an initial shipment to the health department in an amount estimated to be a three months supply. This estimate will be based on the population of the state, weighted by the number of physicians and the number of general, pediatric and maternity hospital beds. Thus, every physician may, if he desires, carry plasma with him, and hospitals may be stocked with a reasonable supply for the treatment of their patients. Replacements of plasma may be obtained on request by physicians or hospitals from the state departments of health or through the local Red Cross chapter, if the department of health does not have a local distribution center. State departments of health in turn will obtain replacements of plasma up to a six months supply by requesting it from the area office of the American Red Cross.

The state departments of health, with the collaboration of the American Red Cross, will undertake to provide appropriate information through their own channels and through state medical societies for the medical profession and the general public in regard to the availability and use of the surplus plasma.

Medical Legislation

MEDICAL BILLS IN CONGRESS

The bill introduced by Representative Priest, Tennessee, H. R. 4512, has been favorably reported to the House of Representatives by the Committee on Interstate and Foreign Commerce to amend the Public Health Service Act to provide for research relating to psychiatric disorders and to aid in the development of more effective methods of prevention, diagnosis and treatment of such disorders.

International Health Organization

The Senate Committee on Education and Labor has reported S. J. Res. 89, with recommendation that it pass, requesting the President, on behalf of the government of the United States, to urge on the United Nations Organization the prompt convening of a conference to draw up the statutes of an International Health Organization.

Miscellaneous

H. R. 4771, introduced by Representative Morrison, Louisiana, provides for the payment of direct federal assistance to individuals permanently and totally disabled, to blind individuals and to certain individuals 65 years of age or over.

A bill introduced by Representative Mills, Arkansas, H. R. 4795, proposes to prevent the smuggling of opium and other narcotic drugs into the United States, particularly from any country where such drugs are permitted to be sold to consumers for nonmedical purposes.

Creation of a Commission on Physical Fitness for American Youth is proposed by H. J. Res. 286, introduced by Representative Weiss, Pennsylvania. This commission, it is contemplated, will investigate the need for the establishment and maintenance of a nationwide physical fitness program and will transmit its recommendations to the President. An appropriation of $50,000 is to be authorized to carry out the purposes of the joint resolution.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Artificial Limb Research.—Research which may yield the first fundamental information on artificial limbs has been started by a team of engineers, medical experts and anthropologists of the University of California. The research will delve primarily into the fundamentals of locomotion of the normal leg and the amputated leg. The scientists hope that improved artificial limbs and operative technics which will make the limb better suited to artificial reconstruction will result. Originally planned exclusively for men in the veterans' age group, the research has been expanded to include men and women in all age groups, since statistics show a far greater number of amputations in the civilian population than among veterans. University faculty members aiding in the research are Howard D. Eberhart, associate professor of engineering, in charge; Alexander S. Levens, associate professor of engineering; Dr. John B. de C. M. Saunders, professor of anatomy; Dr. Verne T. Inman, assistant chemical professor of orthopedic surgery, and Theodore D. McCown, Ph.D., assistant professor of anthropology.

GEORGIA

Cobb Gift of Hospital.—A $250,000 gift from Ty Cobb for the construction of a modern hospital in Royston was reported in the newspapers recently. Mr. Cobb is financing the hospital as a memorial to his parents, who were born and raised in Royston.

Changes in Health Personnel.—Dr. William B. Harrison, medical director of the Northeastern Medical Region, with headquarters in Gainesville, has been named assistant director of the division of maternal and child health of the Georgia state department of health. Dr. Harald M. Graning, San Francisco, has been assigned by the U. S. Public Health Service to serve with the state health department as assistant director of local health organizations. Mr. Thomas F. St. John, recently returned from military service, has joined the state department as information executive in the division of health education.

ILLINOIS

Personal.—Dr. Ernest O. Nay, Terre Haute, Ind., who has been secretary-treasurer of the Wabash Valley Aesculapian Society for nineteen years, was elected president of the society at its recent meeting in Danville. Dr. Harlan A. English, Danville, was elected secretary-treasurer to succeed Dr. Nay. ——Dr. David W. Fey, Peoria, has been appointed fire and police surgeon of Peoria.

Chicago

New Promotions at Northwestern.—Drs. John A. Wolfer and Karl A. Meyer, both associate professors of surgery at Northwestern University Medical School, were recently promoted to professors of surgery. Dr. Wolfer, who graduated at Northwestern University Medical School in 1908, has been a member of the faculty there since 1910. Dr. Meyer, who graduated at the University of Illinois College of Medicine in 1908, has been a member of the faculty at Northwestern since 1926. He is also medical superintendent of Cook County Hospital.

Hematology Research Activities.—The Hematology Research Foundation announces that the Helen Schuman Finnerman Fund, to perpetuate the memory of a leukemia victim, will be used for the hospitalization of special patients in connection with their treatment for blood diseases. A second fund, called the Ruth Reader Fellowship in Hematology, will be used to aid the graduate student working on his Ph.D. and will stimulate interest in blood research. The latter has available $1,500 annually, while the Finnerman Fund will include all monies collected since June 1945. The Hematology Research Foundation, organized in 1944, has a medical advisory council of seven members: Dr. Raphael Isaacs, Michael Reese Hospital, Chicago; Dr. Louis R. Limarzi, assistant professor of medicine, University of Illinois College of Medicine; Dr. Andrew C. Ivy, professor of physiology, Northwestern University Medical School; Dr. Ludvig Hektoen, Chicago Tumor Institute; Dr. Anton J. Carlson, professor emeritus of physiology, University of Chicago School of Medicine; Dr. Italo F. Volini, formerly dean of Loyola University School of Medicine; Dr. Otto Saphir, pathologist, Michael Reese Hospital and University of Illinois. The foundation has 757 members and has raised $16,000 for blood research.

INDIANA

Dearholt Medal Goes to Murray Auerbach.—The Hoyt E. Dearholt Medal of the Mississippi Valley Conference on Tuberculosis was awarded to Mr. Murray A. Auerbach, Indianapolis, for twenty-five years executive secretary of the Indiana Tuberculosis Association. The presentation was made at the Chicago meeting of the conference on October 9.

Personal.—Mr. Robert Yoho, who has been connected with the Indiana State Board of Health, has been appointed director of the division of health and physical education.——Dr. Paul D. Williams has resigned as medical superintendent of the Richmond State Hospital, Richmond. Dr. Otis R. Lynch, Marengo, has been appointed medical superintendent of the Richmond State Hospital to succeed Dr. Paul D. Williams, resigned.

IOWA

Operation of Blood Transfusion Service.—The State University of Iowa College of Medicine, Iowa City, will conduct a course in the operation of blood transfusion and related subjects February 4-March 2 to assist in qualifying physicians to supervise and operate blood transfusion services. The course will be conducted by Drs. Elmer L. DeGowin, assistant professor of internal medicine, and Robert C. Hardin, associate in internal medicine, who assisted Dr. DeGowin in the organization of the blood transfusion service of the University Hospitals. Additional information may be obtained from Dr. John T. McClintock, in charge of postgraduate studies, Medical Laboratories Building, State University of Iowa, Iowa City.

MASSACHUSETTS

George Minot Honored.—Dr. George R. Minot, Nobel laureate in physiology and medicine, 1934, professor of medicine, Harvard Medical School, Boston, and director of the Thorndike Memorial Laboratory of the Boston City Hospital, was honored at a dinner December 5 in recognition of his sixtieth birthday, which he observed December 2. Dr. William B. Castle, Boston, was toastmaster and speakers included Drs. Henry A. Christian, Brookline; Charles Sidney Burwell, Boston; James W. Manary, Boston; Laurence B. Ellis, Boston; Elliott P. Joslin, Boston; James Howard Means, Boston, and Francis M. Rackemann, Boston. Dr. Reginald Fitz, Boston, representing the President and the House of Delegates of the American Medical Association, presented to Dr. Minot its Distinguished Service Medal for his achievements. Dr. Minot was also presented with a bound volume of letters of greetings from numerous scientists throughout the world.

MINNESOTA

Personal.—Drs. Charles Bolsta, Ortonville, and Leonard E. Claydon, Red Wing, recently completed fifty years in the practice of medicine.——Dr. Russell R. Heim, Minneapolis, coroner of Hennepin County, has been reelected secretary-treasurer of the Minnesota Association of Coroners.

Fifty-Five Years of Practice.—Dr. Alfred E. Walker, who was 83 years of age December 3, has been practicing in Duluth continuously since 1890. Dr. Walker and his wife observed the fiftieth anniversary of their marriage last August. He graduated at Bellevue Hospital Medical College, New York, in 1890. *Minnesota Medicine*, which recently credited Dr. Alfred M. Ridgway, Annandale, as being the state's oldest practicing physician (THE JOURNAL, October 27, p. 642), reports that Dr. Walker ties with Dr. Ridgway in age and years of service.

Actions of State Medical Board.—Mrs. Helen Geneva Rudd, Minneapolis, was recently taken to the federal woman's reformatory, Alderson, W. Va., to serve a three year sentence which she had received in 1943. At that time she was placed on probation and ordered to the government hospital at Lexington, Ky., for treatment of drug addiction. The recent revocation of this probation and commitment to the West Virginia institution concludes a twenty year history of a drug addict and represents an activity of the Minnesota State Board of Medical Examiners in ending a long list of impositions of Mrs. Rudd in her attempt to obtain narcotics.

"Dr." John P. LeMay, also known as "Rev." John P. LeMay, and his mother, Mrs. Florence LeMay, were recently sentenced to a term of one year in the Minneapolis Workhouse following their pleas of guilty to the illegal practice of medicine. One witness testified that she had paid a sum of money to the defendants for so-called "detoxivication" treatments, which consisted of an enema which lasted about an hour, "medical pills" and prescription for a diet. When arrested, LeMay had on display in his office the license to practice medicine and the basic science certificate formerly held by a Minneapolis physician who died in January 1944. Testimony revealed that Mrs. LeMay had formerly been employed as secretary of the deceased physician.

NEBRASKA

Personal.—Dr. John M. Thomas, Omaha, has been appointed a member of the Omaha School Board to complete the term of the late Dr. Claude W. Mason, who had been elected to the board for six years, the term to end in 1947.

State Cancer Society Organized.—The Nebraska Society for the Study of Cancer was organized at a meeting in the Paxton Hotel, Omaha, October 26, with Drs. Edward W. Rowe, Lincoln, as president, and Frank H. Tanner, Lincoln, secretary. The society is an outgrowth of a resolution passed by the house of delegates of the Nebraska State Medical Association at its last annual session, which recommended the organization of a cancer research society, the purpose of which will be to cooperate with the American Cancer Society.

NEW JERSEY

Personal.—Dr. Bradford Craver, instructor in pharmacology, University of Rochester School of Medicine and Dentistry, has joined the department of pharmacology, research division, Ciba Pharmaceutical Products, Lafayette Park, Summit.

Library Dedicated to Physician.—Dr. Samuel B. English, for many years medical director and superintendent of the New Jersey Sanatorium for Tuberculous Diseases, Glen Gardner, was honored October 6 when the sanatorium's new library was dedicated to him.

Oil Company Inaugurates Research in Toxicology.—Dr. Joseph P. Holt, formerly associate professor of physiology at the University of Louisville School of Medicine, has joined the medical department of the Standard Oil Company as a specialist in toxicology. He will be responsible for original research in the biologic effects of toxic substances. Dr. Holt will also act as consultant and adviser to the company committee, representing all phases of the company's operation, which is charged with the elimination of potential hazards to workers. With the establishment of this branch of its medical department, Jersey Standard announces that it is the first oil company to undertake its own work in this field. Previously, research has been conducted in outside institutions and laboratories. Dr. Holt graduated at Louisville in 1936. He had been a member of the staff there since 1937.

NEW YORK

Personal.—Dr. Leslie H. Wright has resigned as superintendent of the Genesee Hospital, Rochester.——Dr. Lewis J. Graham, Corning, has been appointed medical director of Corning Glass Works, Corning, filling the vacancy that occurred when Dr. Willis C. Templer died last May.——Dr. Exie E. Welsch, formerly director of the Rochester Guidance Center, has been appointed psychiatrist on the staff of the Bureau of Child Guidance, New York City Board of Education.

New York City

Victory Meeting on Health.—A victory meeting of the Public Health Association of New York City in collaboration with the American Public Health Association was held at the Hotel Pennsylvania, New York, December 13-14. The program was devoted to discussions on environmental sanitation, public health in New York City in 1945, health education, training and recruitment, the war's influence on public health, public health problems highlighted by the war, medical service plans, newer emphasis in public health and what's new in school health.

Annual Report of Research Institute.—The annual report of the Public Health Research Institute of the City of New York, Inc., for the year ended June 30 has recently been made available. The report discusses the activities carried on in the institute during the year and announces that an agreement has been reached between the city of New York and Columbia University providing for a building containing at least 50,000 square feet of laboratory space for the use of the Public Health Research Institute in a proposed research-teaching hospital center. In addition, a 50 bed unit is to be provided in the hospital for use by the institute for the study of disease.

Research Fund Named for Maurice Lewi.—To honor Dr. Maurice J. Lewi on his eighty-eighth birthday, December 1, the Maurice J. Lewi Research Fund, Inc., has been created "to be utilized for research and kindred purposes in connection with the medical sciences, with special attention to as yet unsolved problems in that branch of the healing art known as podiatry (chiropody)." Dr. Lewi graduated at the Albany Medical College in 1877. He has served as professor of medical jurisprudence at the Albany Law School and lecturer at the Albany Medical College. He has also served as president of the first Institute of Podiatry, the Foot Clinics of New York and the Albany County Medical Society. For twenty-one years he was secretary of the New York State Board of Medical Examiners.

Hall of Residence and Alumni Hall.—Two special features figure prominently in the proposed New York University-Bellevue Medical Center, one a Hall of Residence and one an Alumni Hall. The Hall of Residence will cost $750,000 and will comprise ten stories to contain 279 rooms and private living quarters for New York University medical students unable to reside at home who must now find quarters in the scattered boarding houses of the neighborhood. The Alumni Hall will be an auditorium, will cost $500,000 and when completed will be large enough to accommodate all the undergraduates. It will form an individual unit at the heart of the new center and will join the main college and hospital building on one side and the Hall of Residence on another. Campaigns are now under way to raise funds for both units. The entire project with these units and other proposed additions will cost about $27,500,000. The university's share will be $15,000,000. The city will spend $12,500,000 to rebuild its present Bellevue Hospital area, which will join the university's new building along First Avenue. The combined project will cover the nine city blocks between 25th and 34th streets, First Avenue to East River Drive. The university's buildings will occupy a four block area bounded by East River Drive, 30th Street, First Avenue and 34th Street—the section immediately north of the present Bellevue area. The city's new Bellevue development will extend from 25th to 30th streets and from First Avenue to East River Drive. The university section will include a new college of medicine building, a university clinic, a 480 bed university and an Institute of Forensic Medicine, the last mentioned to be the first of its kind in the world and to be built by the city on land to be provided by New York Universiy. Members of the university faculty will staff it. In addition other units will be the Hall of Residence and the Alumni Hall. There will be six institutions housed in New York University structures. One H shaped building to cost $8,500,000 will be nineteen stories and contain the clinic on the ground floor, the college of medicine above that and, superimposed on them both, a fourteen floor hospital.

NORTH DAKOTA

Personal.—Dr. Mary E. W. Soules, Dickinson, was recently appointed director of the southwest district health unit with temporary headquarters in New England. The unit comprises the counties of Slope, Bowman, Golden Valley, Billings, Hettinger and Adams.

Membership of State Medical Board.—Dr. Charles W. Schoregge, Bismarck, is president of the North Dakota State Board of Medical Examiners and Dr. George M. Williamson, Grand Forks, secretary-treasurer and general administrative officer. Other members of the board include Drs. Frederick W. Fergusson, Kulm; Cyril J. Glaspel, Grafton; Olafur W. Johnson, Rugby; William H. Long, Fargo; Archibald D. McCannel, Minot; Frederick L. Wicks, Valley City, and Willard A. Wright, Williston.

OHIO

Personal.—Wirth Howell has been named acting director of the Cleveland Child Health Association. Dr. Richard A. Bolt, Cleveland, resigned as director early this year to assist in the organization of the new school of public health at the University of California, Berkeley (THE JOURNAL, June 30, p. 678).——Dr. Eugene E. Elder, Massillon, has been appointed head of the Youngstown Receiving Hospital for Mental Cases, succeeding Dr. C. Herbert Cronick, who resigned recently (THE JOURNAL, September 29, p. 371).——Dr. George T. Bly-

denburgh, director, student health service, Ohio Wesleyan University, Delaware, is the new secretary-treasurer of the American Student Health Association.——Dr. Jacob B. Moses Sr., Crestline, celebrated his fiftieth anniversary as a practicing physician on July 20.——Dr. and Mrs. Lucius W. Prichard, Ravenna, on October 19 observed their fiftieth wedding anniversary.

Academy of Medicine Lectures.—Lectures to be held during 1946 by the Academy of Medicine of Cincinnati include the following:

Dr. Bruce K. Wiseman, Columbus, January 8, The Spleen and Blood Formation.
Dr. Hiram H. Merritt, Cambridge, Mass., January 22, The Treatment of Epilepsy.
Dr. Milton J. E. Senn, New York, February 5-6 (The B. K. Rachford Lectures, subjects not announced).
Dr. Edmund H. Botterell, Toronto, Canada, February 19, Diagnosis and Treatment of Injuries to the Brain and Spinal Cord.
The Roger Morris Lecture, March 5 (speaker to be announced).
Dr. Sumner L. S. Koch, Chicago, March 19, Injuries of the Nerves and Tendons of the Hand.
Dr. Charles A. Janeway, Boston, April 2, Studies on Clinical Uses of Plasma Protein Fractions.
Dr. Harold G. Wolff, New York, April 16, Reactions of the Nasal Mucous Membranes and the Stomach Engendered by Emotions.
Dr. Frank H. Lahey, Boston, May 7, The Management of Lesions of the Terminal Ileum, Colon and Rectum.
Dr. Walter L. Palmer, Chicago, May 21, The Nature and Treatment of Peptic Ulcers.

OREGON

Personal.—Dr. Dorothea H. Scoville, formerly of New London, Conn., U. S. Public Health Service, has resigned as health officer of Clatsop County to take charge of the maternal and child health work in Richmond, Calif.

New Fellowship in Endocrinology.—The Schering Corporation, Bloomfield, N. J., has created a research fellowship in endocrinology at the University of Oregon Medical School, Portland. The grant of $3,000 for one year's research on gonadotropic hormones is under the direction of Dr. Carl G. Heller, associate professor of physiology and medicine.

PENNSYLVANIA

Philadelphia

First Report of Health Maintenance-Cancer Prevention Clinics.—In the first twelve months of operation of the health maintenance-cancer prevention clinics in Philadelphia, sponsored by the International Cancer Research Foundation, 2,552 persons were examined, 350 of whom were men. Of this total 1,838 were referred to their physicians for care, and 18 who were unable to afford a personal physician were referred to clinics. To obtain a check on the cancer incidence of those patients referred to physicians, letters were sent to 1,148 physicians, all but 91 of whom replied. The reports indicated that 248 examinees failed to see their physicians; 11 of those examined reported a history of cancer in the family. Of malignant disease there were 14 cases, 2 in men (chondrosarcoma of the right ileum and carcinoma of the prostate gland), 12 in women (3 epitheliomas of the skin, 2 carcinomas of the breast, 2 carcinomas of the cervix, 2 carcinomas of the fundus uteri, 1 carcinoma of the gallbladder, 1 carcinoma of the forehead and left tragas and 1 case of abdominal carcinomatosis). Of the many and varied findings may be noted lesions of the cervix 795, cardiovascular diseases 545, skin lesions 406, hemorrhoids 393, benign tumor 253, cystoceles 221, rectoceles 201, nevi 157, genitourinary disturbances 144, obesity 112, hernias 59, prostatic disturbances 55, vaginitis 67, anemias 28, pulmonary tuberculosis 7 and aortic aneurysm 1. Teeth required attention in 102 cases, the feet in 441. Mildred W. S. Schram, Ph.D., secretary of the International Cancer Research Foundation and director of the clinics, has at the request of representative medical groups visited Princeton, N. J., Erie, Pa., New Orleans, Houston, Texas, and some cities in northern California to discuss legal problems and help with the arrangements for establishing such examining centers as those created in Philadelphia (THE JOURNAL, Aug. 26, 1944, p. 248). In her report Dr. Schram states that there is a clinic in Pittsburgh which is almost one year old and that the New Orleans Parish Medical Society has passed a resolution requesting six hospitals to organize clinics. The Massachusetts State Department of Health plans to have one in connection with each of its tumor clinics throughout the state, the first of which was said to be organized in Fall River last spring. The Philadelphia clinics are held at the following hospitals: Hahnemann, Jeanes, Jefferson, Jewish, Lankenau, Pennsylvania, Temple, University of Pennsylvania and Woman's Medical College.

TENNESSEE

Grant for Research.—A grant has been made by Sharp and Dohme, Inc., Glen Olden, Pa., to the department of bacteriology of the University of Tennessee, Knoxville, for a study of sulfonamide drugs in the control of fowl typhoid infection, according to *Science*. The investigation will be conducted by Darlington F. Holtman, Ph.D., professor of bacteriology and head of the department.

New Head of Department of Urology.—Dr. Thomas D. Moore, Memphis, has been appointed professor and head of the department of urology at the University of Tennessee College of Medicine, Memphis, effective December 1. Dr. George R. Livermore, who has been head of the department of urology since 1916, has asked to be relieved of his responsibilities as head of the department. His resignation has been accepted, but he will continue as a professor after a temporary leave of absence.

TEXAS

Dormitory Named for Physician.—The dormitory of Baylor University was recently redecorated and renamed for Dr. John T. Harrington, Waco, since 1932 chairman of the board of trustees at Baylor University. The dormitory will be known as Harrington Hall. Dr. Harrington has practiced medicine sixty-six years and has been associated with Baylor as official physician and as a member of the board of trustees for many years.

Dallas Clinical Conference.—The fifteenth annual spring clinical conference of the Dallas Southern Clinical Society will be held at the Hotel Adolphus, March 18-21, 1946. A preliminary program includes the following speakers:

Dr. Charles A. Doan, Columbus, Ohio, Medicine.
Dr. Jacob A. Bargen, Rochester, Minn., Gastroenterology.
Col. John C. Woodland, M. C., Fort Sam Houston, Military Medicine.
Dr. Alfred Blalock, Baltimore, Surgery.
Dr. John S. Lockwood, New Haven, Conn., Surgery.
Dr. Richard H. Sweet, Boston, Surgery.
Dr. Charles B. Huggins, Chicago, Urology.
Dr. Joe V. Meigs, Boston, Gynecology.
Dr. Donovan J. McCune, New York, Pediatrics.
Dr. Edmund B. Spaeth, Philadelphia, Ophthalmology.
Dr. Albert C. Furstenberg, Ann Arbor, Mich., Otolaryngology.
Dr. William H. Sebrell Jr., Washington, D. C., Basic Science.
Dr. Ira H. Lockwood, Kansas City, Mo., Radiology.

Additional information may be obtained from the secretary of the Clinical Society of the Dallas Southern Clinical Society, 433 Medical Arts Building, Dallas 1, Texas.

GENERAL

National Noise Abatement Council.—At the annual meeting of the National Noise Abatement Council in New York recently Paul J. Washburn, New York, was elected president. Allen Wilson, Chicago, past president of the Greater Chicago Noise Reduction Council, was elected vice president and appointed chairman of a committee to develop a peacetime program designed to reduce needless noise in home, street and factory.

Visitors to the United States.—Two representatives of the Industrial Health Research Board of the British Medical Research Council, Dr. Donald Hunter, physician to the London Hospital and director of its department of research in industrial medicine at the hospital, and Mr. R. S. F. Schilling, secretary, industrial health research board, are studying industrial health activities in the United States. They visited the Council on Industrial Health of the American Medical Association.

Macy Foundation Discontinues Reprint Service.—The War Reprint Service of the Josiah Macy Jr. Foundation will be discontinued by January 1. The reprint service of the foundation has been an effort to bring new and important developments in the science and practice of medicine to medical officers who were largely cut off from the sources of medical information during the war. In the selection of these articles the foundation has had the active cooperation of the committee on pathology of the National Research Council and of the National Committee for Mental Hygiene.

Psychiatric Personnel Placement Service.—Capt. Forrest M. Harrison (MC) has been named director of a newly established psychiatric placement service to function under the auspices of the American Psychiatric Association of the National Committee for Mental Hygiene. The service is designed especially to help physicians and psychiatrists make contacts with training opportunities such as residencies, postgraduate courses and fellowships and to aid institutions in locating suitable candidates for appointments. Physicians interested in psychiatry are invited to send in full biographic state-

ments including personal data, education, training, experience and special desires in order that this service may be of the greatest possible assistance to them. Deans of medical schools, superintendents of hospitals and directors of industrial organizations, clinics, and others employing or participating in the training of psychiatric personnel, are invited to submit full information regarding available positions and courses, including financial details. Foundations, universities and other agencies are asked to report pertinent fellowships in psychiatry, psychosomatic medicine and child guidance. Inquiries should be addressed to Capt. Forrest M. Harrison (MC), National Committee for Mental Hygiene, 1790 Broadway, New York 19.

Insurance Research Fund Starts Operation.—The Life Insurance Medical Research Fund announces that applications for grants may now be made to support fundamental research bearing on cardiovascular disease, including rheumatic fever, hypertension, arteriosclerosis and allied disorders. Funds will become available after January 1, and grants will be made for varying periods of time with the specific requirements of the research problems. Applications for grants may now be made to the chairman of the advisory council and should be transmitted in duplicate through the administrative officer of the institution making application. Requests for grants should include a description of the proposed research, a budget and the date when funds are desired. Applications received by February 1946 will be given consideration at a meeting of the advisory council to be held on or about March 1, 1946. The creation of the research fund was sponsored by the American Life Convention and the Life Insurance Association of America. One hundred and forty-six life insurance companies in the United States and Canada are cooperating in the Life Insurance Medical Research Fund. An advisory council of eight members was created (THE JOURNAL, August 11, p. 1118) and a committee of four physicians was named to the council. Dr. Francis G. Blake, chairman of the advisory council, which will assist the board of directors of the fund in making grants, may be addressed at Yale University School of Medicine, 333 Cedar Street, New Haven 11.

FOREIGN

Manufacture of Penicillin in India.—The government of India has under consideration the appointment of a committee to advise the government on the steps to be taken to manufacture penicillin in India, it is reported.

Baly Medal.—The Baly Medal of the Royal College of Physicians, London, has been awarded to Professor August Krogh of the Zoophysiological Laboratory at Copenhagen, according to *Science*. Dr. Krogh was awarded the Nobel prize in physiology and medicine in 1920.

Personal.—At the request of the Yugoslav Ministry of Health and the army general staff, Sir Harold D. Gillies has recently arrived at the Belgrade airport from England to work with the UNRRA mission. He was to remain in Yugoslavia several days to acquaint local doctors with the latest developments in repair of bodily disfigurements caused by war, accident and disease. He will leave behind several working teams of plastic surgeons trained by him to conduct practical demonstrations in plastic surgery in other areas.

Ernest Sauerbruch Dismissed as Chief of Berlin Health Service.—The four military governors of Berlin voted unanimously October 11 to dismiss Dr. Ernest F. Sauerbruch, Berlin, from his post as chief of the Berlin Public Health Service, the Chicago *Sun* reported. It was stated that Sauerbruch never had been a member of the Nazi party but had been an early admirer of Hitler. The *Sun* stated that the action of the military governing body set something of a precedent. "He prospered under the Nazis and received a position of prominence, whereby his position directly and indirectly contributed to the prestige of the Nazi party." It was indicated that Sauerbruch may still be arrested. Meanwhile his services will be used in a position without administrative authority at the Berlin Charity Hospital, it was stated.

Commission Named to Study Medical Needs in Poland.—Johannes H. Bauer, New York, of the International Health Division of the Rockefeller Foundation, is on loan to the Red Cross to be one of a three member commission to tour Poland to ascertain "prime needs for the $1,000,000 tentatively earmarked for relief work." According to the New York *Times* the UNRRA, which is bringing in food and clothing, has altered the relief problem, shifting the Red Cross emphasis to medical assistance. The *Times* stated that Poland's health and medical service is broken down completely, but the six thousand doctors that the country has left of a prewar total of thirteen thousand are struggling "valiantly" to control disease. It is estimated that 70 per cent of Poland's hospitals and clinics were destroyed. Medical records of the UNRRA show that 12,000 cases of typhus, 22,000 of typhoid, 3,400 of dysentery, 6,000 of scarlet fever and 6,500 of diphtheria were reported in Poland up to the end of August. Last week 32,100 new cases of typhoid alone were reported. Doctors say that there are at least 1,000,000 cases of venereal disease.

Proposed Medical Center at American University of Beirut.—Plans for the construction of a medical center at the American University of Beirut, Lebanon, at the estimated cost of $2,560,000 were announced November 17 by Albert W. Staub, American director of the Near East College Association. The board of trustees of the university has approved the project. The building will have four main wings and two cross wings and will increase the bed capacity by more than 250 per cent, making possible the training of 25 per cent more medical students. As a teaching hospital the building will contain classrooms in conjunction with clinical work, administrative offices, living quarters for interns, staff suites, rooms for the nursing staff and guest rooms. Each service will have its own operating rooms. Nurseries will be designed in the most modern way, with no more than 8 bassinets to a room. Air conditioning will be provided for all operating suites and for private and semiprivate rooms. In the summer, humidity will be controlled throughout the hospital. The American University of Beirut, one of the eight colleges affiliated with the Near East College Association, was founded in 1866 with a charter from the state of New York. Its medical school opened a year later. Work is expected to start on the new building this coming spring.

Government Services

Disposition of Meyerhof's Library

The *Army Medical Library News* announces that collections of the late Dr. Max Meyerhof of Cairo, totaling 10,000 volumes on ophthalmology and ancient Egyptian medicine, were given to the libraries of Fouad I University, Cairo, and Farouk I University, Alexandria. The Army Medical Library has secured from another source an almost complete collection of Dr. Meyerhof's work. This collection includes manuscript materials by Dr. Meyerhof, some of which are unpublished, and also a number of writings about Dr. Meyerhof and his work. A recent photograph of Dr. Meyerhof has been received as a gift of Dr. Henry R. Viets, Boston, to the portrait collection.

Woman Scientists Retire from Public Health Service

Ida A. Bengtson, Ph.D., and Alice C. Evans, Sc.D., were guests of honor at a dinner at Washington recently given in recognition of their retirement after many years of service with the National Institute of Health, U. S. Public Health Service. Dr. Bengtson's retirement will be effective January 1, ending twenty-nine years' service with the institute. Dr. Evans retired October 1, concluding thirty-five years' service. Dr. Evans of Heath, Pa., identified undulant or Mediterranean fever in 1923. Twenty-two years ago, during the course of her laboratory work, she contracted the fever and, although suffering from recurrent attacks, has continued her experiments. By her persistent research she succeeded in tracing the disease to blooded goats which had been imported to the United States from the Mediterranean. Her studies proved also that undulant fever and the disease in cattle in the United States known as Bang's disease are caused by identical germs. Dr. Bengtson of Harvard, Neb., prepared, during 1935-1936, the standard for gas gangrene toxins and antitoxins. This is the standard used by the League of Nation's Health Committee in Copenhagen, Denmark. Following this work, Dr. Bengtson was detailed to study the virus of trachoma at the Missouri Trachoma Hospital. During subsequent research in endemic typhus she became ill with this disease while inoculating rats for study purposes. She contributed to the early studies of the cause and prevention of food poisoning in canned foods, and for the past ten years she has been devoting herself to work in rickettsial diseases.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Nov. 25, 1945.

The Socialist Government and the Medical Profession

The intransigent attitude of the socialist government toward the efforts of its predecessor to come to an agreement with the representatives of the medical profession on the projected national health service seems to be considerably modified. Having made political capital out of the negotiations by denouncing them as "a surrender to the doctors," the present government is now following the same path. By direction of the council of the British Medical Association the secretary, Dr. Charles Hill, wrote to the minister of health, Mr. Bevan, expressing the desire that the representatives of the medical profession should be fully consulted before legislation for a national health service was introduced. Mr. Bevan replied that he would be happy to meet the negotiating committee of the profession before the government finally decides what proposals it will submit to Parliament. At the annual conference of local medical and panel committees, held at the House of the British Medical Association, considerable concern was expressed at the failure of the minister of health to give any indication that he proposed to enter into negotiations with the medical profession before introducing his new health services bill. The reading of his reply to Dr. Hill therefore gave much satisfaction.

Research on Aging

In 1939 a Club for Research on Aging was established in this country. As the name indicates, it consisted of workers engaged in research and was not a medical society in the ordinary sense of the word. There are branches of the club in Britain, the United States, France and Argentina. At present there are only 75 members in the club. The Nuffield Foundation has generously supported a plan for research work, which is being conducted at one of the hospitals of the London County Council. At the end of 1944 Lord Nuffield made a donation of $15,000 to establish an experimental laboratory, the Gerontological Research Unit, which is at present housed in the Departments of Zoology and Physiology of Oxford University. The American branch of the club has already done important work, which includes a museum of senile tissues, clinical research on the effects of vitamins on old persons at Elgin Hospital and the publication of the *Journal of Gerontology*.

Mr. James Sherren: The Surgeon Who Went Back to the Sea

Mr. James Sherren, consulting surgeon to the London Hospital, who has died at the age of 73, had a remarkable career. Born at Weymouth by the sea, he had an inborn love for ships, went to sea and served before the mast. But after a hard and testing experience he suddenly left the sea for the study of medicine at the London Hospital. After a distinguished career he qualified at 27 and was elected to the surgical staff of the hospital in 1902. At this time abdominal surgery was rapidly advancing and he quickly attained a foremost position in this branch. He also did pioneer work with Head on the peripheral nervous system and published an important monograph entitled Injuries of Nerves and Their Treatment. During the war of 1914-1918 he was consulting surgeon to the War Office and did much work in military hospitals. He became a member of the senate of London University and seemed on the way to the highest honors, when he suddenly retired from practice and went back to the sea in the capacity of a ship's medical officer. He was then only 54 and at the height of his career as a surgeon with the possibility of many active years as a leading surgeon before him.

Honor for an American Scientist

The Copley medal of the Royal Society has been awarded to Dr. O. T. Avery for his success in introducing chemical methods in the study of immunity against infective diseases. Dr. Avery, who was elected foreign member of the society last year, is a research fellow of the Rockefeller Institute in New York.

PARIS

(From Our Regular Correspondent)

Nov. 22, 1945.

French Association of Surgery

The forty-eighth Congress of the French Association of Surgery was held in Paris in October, with a large number of French and foreign surgeons attending. This meeting was scheduled to be held in 1939 but had to be postponed because of the war and the German occupation. The present report includes some of the subjects discussed.

THYROTOXICOSIS

For this condition Petit-Dutaillis recommended subtotal thyroidectomy. He reserves medical and physical therapeutic treatment for light, acute forms of the disease and for postoperative relapses. Thyroidectomy should be performed in one or in two stages. He then summarized the 237 cases which he had observed for periods varying from one to nine years. From 80 to 90 per cent of the patients resumed their normal life. Relapses occurred in less than 10 per cent and were ameliorated by radiotherapy. With a series of films, Velti of Paris presented his experience on 5,000 patients with thyrotoxicosis on whom he operated in two stages with an interval of three months. He stressed the importance of technical details. Fontaine and Miss Oswald of Strasbourg reported the 65 thyrotoxic goiters operated on from 1940 to 1944 by subtotal thyroidectomy in a single operation and under local anesthesia. Of 58 patients, 1 is dead. Among 7 other patients operated on in the state of asystolia, there were 2 deaths and 5 recoveries. Arterial tension was transiently modified after the operation; hypertension completely disappeared in only 1 case.

Petit-Dutaillis emphasized the part of thiourea and its derivatives and of aminothiazole in the preparation of serious cases for operation but pointed out the accidents due to this chemotherapy. However, at the recent congress of the French Association for the Advancement of Science Bovet emphasized that the treatment of thyrotoxicosis by aminothiazole was innocuous in 100 cases treated. As for acute postoperative crisis Petit-Duraillis emphasized the part of thyroxin administered immediately after operation. Moor of Brussels believes that thyroxin gives transient results. Delannoy of Lille presented a report on 400 cases with operation in two stages. He practiced the total operation on the right side and the subtotal on the left. He did not operate on 22 seriously ill men, using radiotherapy and medical treatment. He was successful in treating a young woman patient with thyroid grafts after failure following an operation on the sympathetic (Leriche's method). To prevent postoperative myxedema Peycelon of Lyons, as well as Leriche, advised against total thyroidectomy and recommended retaining part of the sound gland. Arnaud of St. Etienne does not remove the isthmus, where, in his opinion, the sound tissue lies.

FUNCTIONAL RESULTS OF BILIODIGESTIVE ANASTOMOSIS

Mallet-Guy of Lyons pointed out that for the last forty years the good results of gallbladder anastomosis have been recorded in France; cholecystogastrostomy was more successful than cholecystoduodenostomy. Bachy and Lenoir presented 7 cases of gastrocholecystostomy for cancer of the pancreas with 2 deaths and 5 survivals. Mallet-Guy dealt with the discussion between the surgeons of Paris and those of Lyons concerning cholecystoduodenostomy in cholelithiasis. The surgeons of Lyons consider it a simplification in serious cases, especially since it leads to a quick recovery, often in eight days. Bachy and Lenoir presented 7 cases of secondary anastomosis, of which 2 were fatal, associated with biliary fistula after external drainage.

ORTHOSTATIC DISEASES AND SURGERY

Constantini of Algiers explained that orthostatic phenomena occur in patients with varicose veins and with cardiac insufficiency. These changes, he believes, are due to a redistribution of blood volume, which, depending on the patient's position, is directed either toward the lower extremities or toward the heart, which then becomes dilated. Clinically there is a sensation of vertigo or anguish with variable pulse and blood pressure. Extensive saphenectomy removes these disorders.

TREATMENT OF HALLUX VALGUS

Vallet of Paris proposed a technic which avoids the pain and the ankylosis: resection of a large part of the phalanx, reduction of the metacarpal head to a cylinder appropriate to the dimensions of the phalanx with a large fibrous interposition.

EXTRAPLEURAL PNEUMOTHORAX

Le Foyer and Delbec of Paris reported 450 cases of extrapleural pneumothorax performed in the surgical treatment of pulmonary tuberculosis over an eight year period. Hemorrhagic or suppurative complications were exceptional.

TREATMENT OF ABSCESSES OF THE BRAIN

Ferey of St. Malo explained Clovis Vincent's technic for ablation of an abscess of the brain. At first all possible antibacterial substances are used and one waits for six to eight weeks, carefully watching the general condition of the patient. The abscess becomes walled off, and then at operation the whole abscess with its capsule is removed in the same way as an aseptic tumor.

NEW TECHNIC OF RENAL SURGERY

Heitz-Boyer of Paris illustrated with films a technic of renal surgery by which complete removal of very large kidneys is possible. He employs a large ileolumbocostal incision and excises the whole of the twelfth rib.

A New Antibacterial Substance: Subtiline

In 1897 Metchnikoff, whose birth centenary was recently commemorated by an exhibition at the Pasteur Institute, found that Bacillus subtilis acts on the tetanic and diphtheritic toxins by destroying their toxic power. On May 8 at the Académie de médecine Jean Ramon, G. Ramon and Richou stated that they have succeeded in determining the antibacterial power of the filtrates of this saprophyte. They prepared a culture medium of vegetable substances with bran as a base. The filtrates obtained are comparatively rich in proteose and have gelatinolytic and bacteriostatic properties. The filtrates are bactericidal and bacteriolytic for the bacillus of diphtheria, the Preisz-Nocard bacillus, the anthrax bacillus, the coccobacillus of pseudotuberculosis and Chiga's dysentery bacillus. The active substance of the filtrate, called subtiline, is now being extracted in a concentrated and purified form with a view to its clinical application.

BRAZIL

(From Our Regular Correspondent)

SÃO PAULO, Nov. 12, 1945.

Intra-Arterial Injection of Penicillin

THE JOURNAL in its July 14 issue published an article on intra-arterial injection of penicillin for infections of the extremities. This method has been widely and successfully employed in São Paulo at the Sanatório São Lucas by Dr. Eurico Branco Ribeiro, according to his article published in August 1944. He proposed the use of penicillin by arterial puncture in infections of the extremities. This method is indicated in acute osteomyelitis, phagedena, panaris and pyogenic arthritis. In order to prolong the therapeutic influence of the penicillin a venous tourniquet can be applied for twenty to thirty minutes. The arterial puncture—in the femoral or in the humeral artery, according to the site of the lesion—can be made once or twice a day for a fortnight without injury to the artery. Dr. Eurico Branco Ribeiro injected 3 to 5 cc. of a solution of penicillin, i. e., 15,000 to 25,000 units of the drug. The injections were well tolerated by the patients. The pain decreased rapidly and healing occurred in a shorter time than with any other treatment. Dr. Eurico Branco Ribeiro emphasizes the safety of the method, its simplicity and the good results obtained.

Cervical Polyps and Polypoid Disorders of the Cervix

Dr. Licinio Dutra was awarded the "Honorio Libero Premium" conferred annually by the Paulista Association of Medicina. In his monograph, Dr. Licinio discusses cervical polyp and polypoid disorders of the cervix. He describes the simple mucous polyp of the cervix and its structural modifications as well as polypoid disorders of the cervix, which macroscopically appear to be simple mucous polyp. He devotes a special chapter to the discussion of gynecologic disorders accompanying cervical polyps which gynecologists should recognize in order to treat them correctly. Dr. Licinio emphasizes the necessity for microscopic examination of all cervical polyps, because of their varied structure. A cervical polyp should be completely extirpated and the site of implantation destroyed by electrocoagulation. If microscopic examination reveals syphilis, tuberculosis, carcinoma or sarcoma the treatment must be correspondingly different. With reference to his own experiences and those of other authors he discusses the problems of early diagnosis of carcinoma of the cervix and squamous metaplasia of a mucous polyp. Dr. Licinio considers the latter a precancerous condition, particularly when associated with irritant agents.

Pemphigus Foliaceus

A large new pavilion for women has been opened at the Hospital for Pemphigus Foliaceus ("wildfire"), a disease prevalent in the interior of the state of São Paulo. Dr. João P. Vieira, director of the special service of pemphigus foliaceus, reported that the hospital capacity has been increased from 60 to 150 beds, and many improvements have been introduced which will help in the campaign against this dreadful disease.

The Association of Thiamine Hydrochloride and Mono-Io-Phenil

The object of Dr. Cândido Botafogo's recent experimental work was to ascertain whether thiamine hydrochloride undergoes alteration in the presence of mono-io-phenil, a salt composed of organic iodine and aminopyrine, or whether it retains all its biologic properties. The possibility of the oxidation of thiamine hydrochloride and its transformation into thiochrome, rendering it inactive in the treatment of multiple neuritis, is well known. The tests carried out by the author were based on the cure of polyneuritis in pigeons (Kinnersley-Peters) and in

rats (M. J. Smith). Dr. Cândido describes the principal technic in the processes mentioned. Some animals with induced thiamine hydrochloride deficiency were injected with the substance to be tested and others received the control substance. It was found that with the two solutions an identical response was obtained from the animals under observation. Dr. Cândido Botafogo concludes that thiamine hydrochloride retains its properties satisfactorily in the presence of mono-io-phenil when present in the dose indicated in the solution tested.

Brief Items

Dr. Samuel Leão de Moura, director of the Public Health Laboratory of the city of Santos, São Paulo, has recently published a report concerning schistosomiasis mansoni discovered by him in the district of Saboó. Dr. Moura has examined the feces of 1,126 persons of this district and found 113 positive cases (9.1 per cent). The disease until now has not occurred in the city and probably was imported from northeastern Brazil through commercial exchanges between the main port of the state and that region, where the infection is highly prevalent. The drastic measures that have been taken to control the spread of the disease have been fully approved by Dr. Cesar Pinto, a specialist of the Oswaldo Cruz Institute of Rio de Janeiro, who has just visited the city of Santos.

This month at São Paulo the Paulista Association for the Study of Industrial Medicine was organized. Dr. Borges Vieira, professor at the School of Public Health of the University of São Paulo, was elected president of the society.

Dr. Antonio C. Camargo, professor emeritus of medicine at the University of São Paulo, recently addressed the Paulista Medical Association on the "Councils of Medicine" created by a recent decree of President Getutulio Vargas. Dr. Camargo reminded the association that these councils would have represented an improvement in the practice of medicine in Brazil if the decree had followed the main lines of the bill introduced in 1937 in the Brazilian congress at the request of the leading medical societies. Nevertheless the decree of President Vargas has treated the subject merely as a punitive measure which was expected to be used as a political weapon against physicians at a time when Brazil is hoping to have fair elections for president and for congress. As an old and mature practitioner of medicine, Dr. Camargo declared that he was against the organization created by the Vargas decree.

Personal

Dr. Italo Sciascia, associate professor of medicine at the Paulista School of Medicine, has left for the United States at the invitation of Dr. Thomas I. Price of New York to work for some time at one of the hospitals of that city.

The São Paulo Association of Medicine and the São Paulo chapter of the Brazilian College of Surgeons have recently held a joint meeting to honor the memory of Dr. Sergio Paiva Meira, anatomist and surgeon, who was one of the leading professors at the University of São Paulo. At this meeting Dr. Sebastião Hermeto, Dr. Eurico S. Bastos and Dr. Orlando Aidar spoke in praise of the medical achievements of the late Professor Meira.

Dr. João Bicudo Junior, associate professor of dermatology at the University of São Paulo and past president of the Section of Dermatology of the Paulista Medical Association, has been appointed head of the outpatient department of the Division of Dermatology and Syphilology of the Santa Casa Hospital. Dr. Bicudo succeeds Dr. Aguiar Pupo, a leading dermatologist of this city.

Dr. Ciro Rezende, ophthalmologist, of São Paulo, has been elected member of the local chapter of the Brazilian College of Surgeons. Dr. Rezende is associate professor of ophthalmology at the University of São Paulo.

SWITZERLAND

(From Our Regular Correspondent)

GENEVA, Nov. 14, 1945.

Mental Hygiene in Switzerland

There is a great increase in interest in Switzerland toward all questions of mental hygiene. In many towns, offices called "Offices médicaux pédagogiques" have been created to assist the "problem child." A series of lectures is given annually by the Institute of the Sciences of Education of the University of Geneva. The last series was specially concerned with the problem of intelligence. Dr. Brantmay spoke on the importance of intelligence and how great a problem it is in mental hygiene. Dr. F. Rilliet, the Geneva school doctor, spoke on the illnesses that could endanger the intellectual development of children. Professor G. de Morsier gave a lecture on "intelligence and the brain" and Professor F. Morel, director of the psychiatric clinic of Geneva, spoke on intelligence in psychiatry.

Swiss Physicians for the Current Year

Medical students in Switzerland at present total 2,608. Of these 2,295 are Swiss and 313 are foreigners. Students who have just graduated number 233, of whom 34 were women. The total number of Swiss doctors is 4,073, to which can be added 1,443 assistants. There is 1 physician for 1,171 inhabitants. In a town it amounts to 1 physician for 668 inhabitants. Of the total number of physicians 1,634 are specialists. There are now 142 physicians in practice who are between 71 and 75 years of age, 66 physicians who are between 76 and 80 years of age, and 26 who are more than 81. In 1938 the figures were 100, 38 and 16 respectively for these older age groups.

Medical Consultative Commission of International Red Cross

The Medical Consultative Commission of the International Red Cross Committee, composed of physicians and one nurse, was created in 1943 with the aim of centralizing medical data coming from all countries, particularly those concerning the medicaments and sanitary material. It also provided help of medical or prophylactic character to the officers of prisoner of war or civil internee camps. From numerous national organizations this commission receives important documentation on the health conditions of the world population, on the different illnesses and on the development of epidemics. It encourages the numerous national Red Cross organizations to create qualified medical personnel. It has organized special courses for Swiss physicians on the prophylaxis and the campaign against numerous epidemics. In Switzerland 198 physicians, 211 nurses and 69 technicians are at the disposal of the commission. Thus a specialized personnel from a neutral country can start immediately to an epidemic area, collect the necessary data, give prompt advice as to what methods of help can be used and transmit as quickly as possible the necessary material from the stocks of the International Red Cross Committee.

Marriages

ARNOLD JOSEPH CERASOLI, New York, to Miss Concettina Yolanda Magri of Mount Vernon, N. Y., November 10.

JAMES T. CARROLL, Fulton, N. Y., to Miss Lucille Jeannette Butter of Richfield, N. J., in Bogota, N. J., August 25.

ROBERT CALVIN BROWNLEE JR., Due West, S. C., to Miss Jewel Pharr in Charlotte, N. C., October 18.

SELMAR BURCHART to Miss Katherine Hawks, both of Memphis, Tenn., October 10.

IRVING CRAMER, Utica, N. Y., to Miss Helen Rubin of Albany, October 6.

Deaths

Charles Fletcher Davis ✠ Colonel, M. C., U. S. Army, retired, Parsons, Kan.; born in Arlington, Ore., May 20, 1888; University of Oklahoma School of Medicine, Oklahoma City, 1914; Army Medical School, Advanced Course, in 1932, and Medical Field Service School, Advanced Course, in 1936; fellow of the American College of Surgeons and the American Public Health Association; received the degree of master of public health at Johns Hopkins University School of Hygiene and Public Health in 1940; served overseas during World War I; entered the medical corps of the U. S. Army as a first lieutenant in 1920; rose through the various grades to that of colonel; trained more than twenty general and other hospitals for overseas duty in the various theaters during World War II and served overseas in North Africa as the commanding officer of the 21st General Hospital; retired Jan. 1, 1945 after having served twenty-five years in hospitals of the U. S. Army; superintendent of the Parsons State Hospital; died suddenly, July 26, aged 57, of coronary thrombosis.

James Wilkinson Jervey ✠ Greenville, S. C.; Medical College of the State of South Carolina, Charleston, 1897; born in Charleston, S. C., Oct. 19, 1874; honorary member and twice president of the South Carolina Medical Association; president of the South Carolina Ophthalmological and Otolaryngological Society, 1922-1923; past president of the Greenville County Medical Society; president of the American Laryngological, Rhinological and Otological Society, 1933-1934, serving twice as chairman of its southern section; served as chairman of the section on ophthalmology and otolaryngology, for many years on the council and in 1937-1938 as president of the Southern Medical Association; member of the American Ophthalmological Society; specialist certified by the American Board of Ophthalmology and the American Board of Otolaryngology; medical director of Dr. Jervey's Private Hospital; formerly editor of the *Journal of the Medical Association of South Carolina;* died November 1, aged 71.

Robert Henry Ferguson ✠ Orange, N. J.; University of Vermont College of Medicine, Burlington, 1898; born in Boston, Nov. 4, 1857; member of the Massachusetts Medical Society and the American Association for the Advancement of Science; fellow of the American Society of Anesthetists, New York Society of Anesthetists, Interstate Association of Anesthetists, Pacific Coast Association of Anesthetists, Society of Anesthetists of Great Britain, Canadian Society of Anesthetists, Royal Society of Medicine, London, England, and the New York Academy of Medicine; associate member of the Southern Association of Anesthetists; served as chief of the medical department and the department of anesthetics, E. R. Squibb & Sons; devised an inhaler for etherization by the open drop method; died August 30, aged 87, of myocarditis.

James Philip Boylan ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1927; born in Brooklyn, Sept. 1, 1895; assistant clinical professor of obstetrics and gynecology at the New York Medical College, Flower and Fifth Avenue hospitals; diplomate of the National Board of Medical Examiners; specialist certified by the American Board of Obstetrics and Gynecology, Inc.; fellow of the American College of Surgeons; served during World War I; on the staffs of the New York City Hospital, House of Detention for Women, Vanderbilt Clinic and St. Clare's Hospital, where he died September 25, aged 50, of carcinoma of the larynx.

Mills Sturtevant ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1908; born in Whitfield, N. H., April 7, 1882; professor of clinical medicine at the New York University College of Medicine; member of the American Gastro-Enterological Association and the International Gastro-Enterological Association; fellow of the American College of Physicians; specialist certified by the American Board of Internal Medicine; served on the staff of the Bellevue Hospital and the Rockaway Beach (N. Y.) Hospital; died in the Harkness Pavilion, Columbia-Presbyterian Medical Center, October 29, aged 63, of pneumonia.

Clifford Evan Howard, Ogdensburg, N. Y.; Queen's University Faculty of Medicine, Kingston, Ont., Canada, 1928; born in Gananoque, Ont., in 1905; member of the American Psychiatric Association; interned at the Royal Victoria Hospital in Montreal, Que.; formerly staff physician and surgeon at St. Lawrence Lumber Company in Trinity Bay, Que.; a member of the psychiatric staff of the Binghamton State Hospital in Binghamton from 1932 to 1942, when he became clinical director at the St. Lawrence State Hospital, a position he held at the time of his death on August 10, aged 40, of uremia.

Charles Lee Beeching ✠ Surgeon, Lieutenant Commander, U. S. Navy, retired, Kansas City, Mo.; University Medical College of Kansas City, 1908; entered the U. S. Navy on Oct. 3, 1911; retired June 30, 1937; recalled to active duty 1940, later retired because of illness; died August 29, aged 63, of coronary occlusion and hypertrophic arthritis.

William C. Boteler, Minneapolis; University of Maryland School of Medicine, Baltimore, 1878; died September 17, aged 90, of bronchopneumonia, arteriosclerosis and encephalopathy.

Robert Lester Botkin ✠ Duquesne, Pa.; University of Pittsburgh School of Medicine, 1919; director of the city bank; died September 24, aged 50, of coronary occlusion.

Oliver J. Caza ✠ Skowhegan, Maine; M.B. in 1905 and M.D. in 1907, School of Medicine and Surgery of Montreal, Faculty of Medicine of the University of Laval at Montreal; past president of the Somerset County Medical Society; served as town and school physician; on the staff of the Redington Memorial Hospital; for twenty-five years house physician for the State Reformatory for Women; died in the New England Baptist Hospital, Boston, October 25, aged 64, following an operation for carcinoma.

John Grigsby Clem, Louisville, Ky.; Hospital College of Medicine, Louisville, 1903; member of the American Medical Association; served during World War I; died in St. Joseph Infirmary September 28, aged 65, of coronary occlusion.

Frederick Stephens Cole, Inlet, N. Y.; College of Physicians and Surgeons, medical department of Columbia College, New York, 1890; health officer; formerly member of the state legislature; served during World War I; died in Buffalo September 12, aged 80, of carcinoma of the bladder.

John Joseph Connolly, Louisville, Ky.; University of Louisville Medical Department, 1906; served as coroner and deputy coroner; died in SS Mary and Elizabeth Hospital September 11, aged 73, of hypertension.

Charles Johnston Davis ✠ Deerfield, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1902; served in the medical corps of the U. S. Army during World War I; formerly health commissioner; on the staff of the Highland Park (Ill.) Hospital; president of the Deerfield State Bank; died September 15, aged 70, of hypertension and hemiplegia.

Raymond del Mas, Hugo, Minn.; Dunham Medical College, Chicago, 1901; Hering Medical College, Chicago, 1903; died in San Francisco September 16, aged 74.

Charles W. Denny, North Madison, Ind.; University of Louisville (Ky.) Medical Department, 1893; member of the American Medical Association; honorary member of the Indiana State Medical Association; on the staff of the King's Daughters' Hospital in Madison, where he died September 26, aged 77, of coronary thrombosis and diabetes mellitus.

James Louis Early ✠ Radford, Va.; University College of Medicine, Richmond, 1901; past president of the Southwestern Virginia Medical Association; member of the American Medical Association; died September 1, aged 68.

John Thomas Eckert, Allentown, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1903; member of the American Medical Association; on the staff of the Sacred Heart Hospital, where he died September 13, aged 68, of generalized carcinomatosis.

Robert M. Elliott, Canandaigua, N. Y.; University of Buffalo School of Medicine, 1890; member of the American Medical Association and the American Psychiatric Association; formerly associate clinical professor of mental diseases at the Long Island College Hospital; for many years superintendent of the Willard (N. Y.) State Hospital; formerly junior assistant physician at the Rochester State Hospital; at one time affiliated with the Brooklyn State Hospital; died October 5, aged 82, of coronary thrombosis.

Toivo Johannes Forsstrom, Astoria, Ore.; Helsingfors Universitet Medicinska Fakulteten, Helsingfors, Finland, 1897; died August 16, aged 78.

Frank Durkee German ✠ Pontiac, Mich.; Detroit College of Medicine, 1906; served during World War I; examining physician for the draft board during World War II; on the staff of the Pontiac General Hospital; died September 26, aged 65, of carcinoma of the rectum.

Joseph Shem Gian-Franceschi ✠ Buffalo; University of Buffalo School of Medicine, 1908; specialist certified by the American Board of Radiology, Inc.; member of the Radiological Society of North America, Inc., and the American College of Radiology; secretary and past president of the Buffalo Radiological Society; member of the staffs of the Emergency and Columbus hospitals; an associate staff member of the Lafayette General Hospital; died October 17, aged 61, of cerebral hemorrhage and chronic nephritis.

Carlyle Newton Haines ✠ Athens, Pa.; College of Physicians and Surgeons, Baltimore, 1908; specialist certified by the American Board of Urology, Inc.; past president of the Bradford County Medical Society; member of the American Urological Association; fellow of the American College of Surgeons; served during World War I; formerly associated with the Guthrie Clinic and for many years on the staff of the Robert Packer Hospital in Sayre; died September 22, aged 59, of coronary occlusion.

William Joseph Harkins, Quincy, Mass.; University of Vermont College of Medicine, Burlington, 1911; member of the American Medical Association and the New England Otological and Laryngological Society; served overseas during World War I; for many years on the staffs of the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary in Boston; served as consultant at the Quincy City, Weymouth (Mass.), Milton (Mass.) and other hospitals; died August 25, aged 59.

Grace Frith Hagans Jerger, Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1904; died September 18, aged 66.

Audley Clark Joslyn, Portland, Ore.; University of Oregon Medical School, Portland, 1928; captain, medical reserve corps, U. S. Army, not on active duty; died August 29, aged 49, of pulmonary hypertension with heart disease.

Henry Randal Kenny, Chicago; University of Dublin School of Physic, Trinity College, Dublin, 1909; member of the American Medical Association; on the staffs of the Illinois Masonic Hospital, Columbus Hospital and the Alexian Brothers' Hospital, where he died September 9, aged 59.

Frederick C. Liddle ✠ Dorset, Vt.; University of Vermont College of Medicine, Burlington, 1887; honorary member of the Vermont Medical Society; past president and vice president of the Bennington County Medical Society; honorary president of the Dorset Memorial Library; died September 17, aged 84, of pneumonia following a fractured rib received in a fall.

Edward Frank Marsh ✠ White Plains, N. Y.; Albany (N. Y.) Medical College, 1882; died September 4, aged 86, of carcinoma of the stomach.

Walter Bruce Maxwell, Nesbitt, Miss.; Louisville (Ky.) Medical College, 1874; for many years postmaster; died September 20, aged 93.

Bernard Vincent McCabe, Helena, Mont.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1908; served as president of the Lewis and Clark County Medical Society; died in St. John's Hospital in August, aged 73.

James McClymonds McGeorge, Salem, Ohio; University of Wooster Medical Department, Cleveland, 1902; member of the American Medical Association; fellow of the American College of Surgeons; served during World War I; on the staff of the Salem City Hospital, where he died September 1, aged 70, of generalized arteriosclerosis.

John Dickinson Milburn, East Hampton, Conn.; the Hahnemann Medical College and Hospital, Chicago, 1910; served as health officer and medical examiner; on the staff of the Middlesex Hospital, Middletown; died September 17, aged 64, of coronary thrombosis.

Willis Moore, Vermont, Ill.; Chicago Medical College, 1890; died August 16, aged 82.

I. J. Morris, Dallas, Texas; Vanderbilt University School of Medicine, Nashville, Tenn., 1905; died in Chicago August 18, aged 63.

Robert Shibley Moth, Council Bluffs, Iowa; the Hahnemann Medical College and Hospital, Chicago, 1905; served overseas during World War I; on the staff of the Jennie Edmundson Hospital, where he died August 27, aged 63, of carcinoma of the colon.

James Kay Newman, Omaha; Universidad Nacional Facultad de Medicina, Mexico, D. F., 1879; member of the American Medical Association; honorary member of the Nebraska State Medical Association; died in St. Joseph Hospital October 5, aged 96, of bronchogenic carcinoma.

Lewis Beauregard Newsom, West Monroe, La.; Memphis (Tenn.) Hospital Medical College, 1891; member of the American Medical Association; served on the staff of St. Francis Sanitarium, Monroe; died in Shreveport recently, aged 83, of senility.

Francis LeJau Parker ✠ Charleston, S. C.; Medical College of the State of South Carolina, Charleston, 1909; professor of chemistry at his alma mater, where he had served for a period as dean; died in Hendersonville, N. C., August 20, aged 69, of coronary disease.

Herman Frederick Schrader, Browning, Mont.; Jefferson Medical College of Philadelphia, 1910; member of the American Medical Association; served as vice president of the North Central District Medical Association; city health officer; affiliated with the Indian Service and physician in charge of the Blackfeet Hospital; died in the Deaconess Hospital, Great Falls, August 23, aged 65, of heart disease and cerebral hemorrhage.

John King B. E. Seegar Sr., Baltimore; Baltimore Medical College, 1900; member of the American Medical Association; fellow of the American College of Surgeons; gynecologist, St. Agnes' and Bon Secours hospitals; associate in gynecology at the Maryland General Hospital; died in the Johns Hopkins Hospital August 24, aged 76, of carcinoma of the stomach.

Gustav Maxim Steinbach ✠ Brooklyn; University of Aberdeen Faculty of Medicine, Scotland, 1933; interned at the Montefiore Hospital for Chronic Diseases in New York; served a residency and as a member of the staff of the Jewish Hospital; died August 30, aged 42, of coronary disease.

Claude Earl Stump, Kansas City, Mo.; University of Kansas School of Medicine, Lawrence and Kansas City, 1906; served as police surgeon and on the staff of the Ralph Sanitarium; died August 26, aged 63.

John Francis Swarens, Kansas City, Mo.; National Normal University College of Medicine, Lebanon, Ohio, 1892; Kentucky University Medical Department, Louisville, 1899; Southwestern Homeopathic Medical College and Hospital, Louisville, Ky., 1900; died August 23, aged 78, of coronary occlusion.

Alfred Charles Taylor, Mona, W. Va.; Chicago College of Medicine and Surgery, 1916; member of the American Medical Association; died in a hospital at Morgantown October 25, aged 67, of cerebral hemorrhage.

Frederick William Taylor, Baltimore; University of Maryland School of Medicine, Baltimore, 1884; formerly society editor of the *Sunday Sun;* died in the Johns Hopkins Hospital August 16, aged 83.

Frank C. Tinsley, Indianapolis; Central College of Physicians and Surgeons, Indianapolis, 1891; on the staff of the Methodist Hospital; died August 23, aged 81, of septic cystitis.

Newton Murray Tucker, Nashville, Tenn.; University of Tennessee Medical Department, Nashville, 1898; veteran of the Spanish-American War; died August 15, aged 74, of heart disease.

Smith Lanier Turner, Williston, Fla.; Atlanta College of Physicians and Surgeons, 1911; health officer of Levy County; died in Ocala, August 2, aged 59, of acute dilatation of the heart following an operation for gangrenous gallbladder.

George G. Van Mater, Peru, Ind.; New York Homeopathic Medical College and Hospital, New York, 1892; served during World War I; died in the Dukes-Miami County Memorial Hospital August 28, aged 82, of carcinoma.

Oscar Clinton Wainscott, Peru, Ind.; University of Louisville (Ky.) Medical Department, 1893; Bellevue Hospital Medical College, New York, 1896; served as medical examiner for the Miami County Draft Board during World War I; formerly president and chairman of the board of directors of the Wabash Valley Trust Company; died September 3, aged 75, of carcinoma of the prostate.

Edwin Bernard Weldon ✠ Bridgeport, Conn.; College of Physicians and Surgeons, Baltimore, 1913; specialist certified by the American Board of Pediatrics, Inc.; member of the American Academy of Pediatrics; attending pediatrician at Englewood Hospital and St. Vincent's Hospital, where he died August 11, aged 54, of coronary thrombosis.

Paul Whelan ✠ Seattle; University of Pennsylvania School of Medicine, Philadelphia, 1915; on the staffs of the King County and Doctors hospitals; died August 28, aged 58, of carcinoma of the stomach.

William Shipman Wiggins ✠ Exeter, Neb.; Michigan College of Medicine and Surgery, Detroit, 1895; past president of the Fillmore County Medical Society; for many years president of the board of education in Exeter and surgeon for the Burlington and the Chicago and Northwestern railroads; on the staff of the Lutheran Hospital in York; died August 19, aged 75.

Alexander E. Wilcox, Adrian, Mich.; Saginaw Valley Medical College, Saginaw, 1900; died August 3, aged 75.

William Lewis Yeomans, Bucyrus, Ohio; University of Toronto Faculty of Medicine, Toronto, Ont., Canada, 1897; member of the American Medical Association; on the staff of the Bucyrus City Hospital; died August 30, aged 71, of arteriosclerosis.

Jerome Edward Young, Troy, N. Y.; Albany Medical College, 1896; died in the Troy Hospital August 10, aged 71, of pulmonary edema and chronic myocarditis.

DIED WHILE IN MILITARY SERVICE

Lawrence Benjamin ✠ Chicago; Loyola University School of Medicine, Chicago, 1929; interned at St. Francis Hospital; commissioned a captain in the medical corps, Army of the United States, on Nov. 17, 1943; died Nov. 27, 1944, aged 44.

Richard Booth Jr., Lynchburg, Va.; University of Virginia Department of Medicine, Charlottesville, 1943; interned at the Virginia Mason Hospital in Seattle; began active duty as a first lieutenant in the medical corps, Army of the United States, on Jan. 2, 1944; died in Germany May 12, aged 28, of injuries received in an airplane crash.

Arthur Smith Dutton, Indiana, Pa.; Western Reserve University School of Medicine, Cleveland, 1938; member of the American Medical Association and the Ohio State Medical Association; interned and served as assistant resident in surgery at Cleveland City Hospital; began active duty as a first lieutenant in the medical corps, Army of the United States, in March 1941; served as executive officer of the Station Hospital, Fort Knox, Ky.; promoted to captain and major; in August 1943 went to North Africa with the 118th Field Hospital; promoted to lieutenant colonel; later transferred to the 24th General Hospital and stationed in Italy; died in the 64th General Hospital, in northern Italy, July 17, aged 37, of exfoliative dermatitis and acute nephritis.

Irving Fagin, New York; St. Louis University School of Medicine, 1939; diplomate of the National Board of Medical Examiners; interned at the Beth David Hospital; began active duty on June 26, 1941, as a first lieutenant in the medical corps, Army of the United States; promoted to captain; died in the Southwest Pacific area Sept. 18, 1944, aged 28.

William Duncan Frostic, Wyandotte, Mich.; University of Michigan Medical School, Ann Arbor, 1937; member of the American Medical Association; formerly an intern and resident in surgery at the Harper Hospital in Detroit; served a residency in medicine at the Pontiac General Hospital in Pontiac; began active duty as a first lieutenant in the medical corps, Army of the United States, on Oct. 24, 1941; promoted to captain on June 21, 1943; flight surgeon with the American Air Forces; overseas since March 1945; killed July 30, aged 34, when his plane crashed while on a routine flight from a U. S. Army Base at Oahu, Hawaii.

William Hildreth Gillespie, New York; Columbia University College of Physicians and Surgeons, New York, 1938; diplomate of the National Board of Medical Examiners; served an internship, assistant residency and fellowship at the Presbyterian Hospital; began active duty as a captain in the medical corps, Army of the United States, on Feb. 15, 1942; promoted to major in September 1944; served in the United States and overseas in the Belgian Congo, Casablanca, Oran and France, with the Presbyterian Hospital Unit, the 23d Station Hospital, the 237th General Hospital and the 23d General Hospital; died in France June 7, aged 32, of acute infectious hepatitis with jaundice.

Thomas Herbert Greenway ✠ Three Rivers, Mass.; Tufts College Medical School, Boston, 1927; interned at the Eastern Maine General Hospital in Bangor, Maine; served as a member of the board of health and as associate medical examiner; member of the staff of the Wing Memorial Hospital in Palmer; major, medical corps, Army of the United States; shortly after entering the service was sent to the South Pacific as medical officer with an amphibious engineers' unit; at Lae was named for conspicuous heroism; died in the Cushing General Hospital, Framingham, June 23, aged 44, of carcinoma of the rectum with metastasis to the liver.

Leonard Watson Hassett ✠ Colonel, M. C., U. S. Army, Washington, D. C.; Tufts College Medical School, Boston, 1910; Army Medical School in 1922; became a lieutenant in the National Guard in 1912 and served as a captain during World War I; entered the medical corps of the U. S. Army as a captain on July 1, 1920; rose through the various grades to that of colonel; commanding officer of the Station Hospital, Camp Pickett, Blackstone, Va.; fellow of the American College of Surgeons; at one time served on the faculty of the University of Vermont College of Medicine in Burlington; died in the Woodrow Wilson General Hospital, Staunton, Va., July 25, aged 58, of coronary thrombosis.

Charles George Hutter ✠ Colonel, M. C., U. S. Army, Santa Barbara, Calif.; Northwestern University Medical School, Chicago, 1913; fellow of the American College of Surgeons; Army Medical School in 1916 and the advanced course at the Medical Field Service School in 1934; entered the medical reserve corps of the U. S. Army as a first lieutenant in March 1916; vacated in June 1916 and entered the medical corps of the regular army in the same rank; served with the third division overseas during World War I; promoted through the various ranks to that of colonel; formerly liaison officer, Office of the Surgeon General, U. S. Army, to the American Medical Association; died in the Hoff General Hospital June 26, aged 59, of hypopharyngeal hemorrhage due to carcinoma of the hypopharynx.

Edgar Shugert Ingraham, Mercer, Pa.; Western Reserve University School of Medicine, Cleveland, 1935; member of the American Medical Association and the American Association of Pathologists and Bacteriologists; specialist certified by the American Board of Pathology, Inc.; interned at the Presbyterian Hospital in Philadelphia; interned and served a residency at the University Hospitals in Cleveland; commissioned a first lieutenant in the medical corps, Army of the United States, on Sept. 10, 1941; promoted to captain on Nov. 9, 1942 and major on July 26, 1944; chief of laboratory services in the 142d Base Hospital at Calcutta, India; killed in India August 6, aged 34, in an airplane crash.

Levy Stephen Johnson ✠ Colonel, M. C., U. S. Army, Washington, D. C.; University of Texas School of Medicine, Galveston, 1912; Army Medical School in 1918 and postgraduate course in 1935; School for Flight Surgeons in 1920; began active duty as a first lieutenant in the medical corps of the U. S. Army on June 28, 1917; rose through the various grades to that of lieutenant colonel on July 28, 1937; promoted to the temporary rank of colonel on Oct. 31, 1942; died in Camp Barkeley, Texas, Jan. 8, 1943, aged 56, of heart disease.

Richard Paul Morden, Jefferson, Iowa; State University of Iowa College of Medicine, Iowa City, 1934; member of the American Medical Association; interned at the Mercy Hospital in Des Moines; commissioned a first lieutenant in the medical corps, Army of the United States, on July 30, 1942; promoted to captain; arrived in England in June 1943; died near Cambridgeshire, England, May 18, aged 39, of subarachnoid hemorrhage.

Robert John Rosenheimer, Kewaskum, Wis.; Marquette University School of Medicine, Milwaukee, 1943; interned at the Milwaukee County Hospital, where he served a residency in pathology; commissioned a first lieutenant in the medical corps, Army of the United States, in 1944; died in a hospital at Washington, D. C., July 16, aged 28, of tumor, argentaffine type, malignant, primary in the cecum, with metastasis to the bladder and ureters.

Henry Sutherland-Campbell, Los Angeles; McGill University Faculty of Medicine, Montreal, Que., Canada, 1920; associate clinical professor of medicine (dermatology) at the University of Southern California School of Medicine; member of the American Medical Association; specialist certified by the American Board of Dermatology and Syphilology; on the staffs of the Good Samaritan and St. Vincent's hospitals; commissioned a major in the medical corps, Army of the United States, on Sept. 8, 1942; died July 7, aged 55.

Robert Emery Trekell, Wellington, Kan.; University of Kansas School of Medicine, Lawrence and Kansas City, 1944; an acting assistant surgeon lieutenant (jg) in the U. S. Navy while he served an internship at the Naval Hospital in Farragut, Idaho; lieutenant (jg) in the medical corps, U. S. Naval Reserve; died in the Pacific area April 22, aged 25, of accidental acute therapeutic poisoning.

Thomas Loftin Weber ✠ Olney, Ill.; Washington University School of Medicine, St. Louis, 1937; interned at the University Hospital in Baltimore and served a residency in pediatrics at St. Louis Children's Hospital in St. Louis; began active duty as a first lieutenant in the medical corps, Army of the United States, on Nov. 12, 1942; promoted to captain on Feb. 5, 1944; accidentally killed September 4, aged 34, while making a routine parachute jump at the Army Air Base at Watertown, S. D.

Correspondence

INJECTIONS OF DISTILLED WATER

To the Editor:—I should like to comment on your answer to an inquiry regarding the usefulness of injections of distilled water (THE JOURNAL, Oct. 20, 1945, p. 584).

You state that "there appears to be no basis for preference for water over isotonic solution of sodium chloride." I can think of at least three conditions in which this statement would not apply and, indeed, might even lead to danger. First, water is preferable in any condition in which there is deprivation of water alone, e. g., for a patient who is unable to replace water lost as insensible perspiration and from the lungs. This leads to dehydration, which involves some loss of electrolyte, but in general is associated with a retention of salt, or hypertonicity of the body fluids. True replacement therapy in such a case would consist in the injection of water with only a small amount of electrolyte. For such purposes a hypotonic solution of sodium chloride, preferably in 5 or 10 per cent glucose solution, should be employed. While it is true that an isotonic saline solution might correct dehydration in such a patient, it would be necessary to provide for the excretion of the excess salt; otherwise the excess might easily provoke deleterious results. For example, if the patient needed 3 liters of water and, say, 5 Gm. of sodium chloride, the use of isotonic saline solution would introduce 27 Gm. of sodium chloride, of which 22 is excessive and which would provoke difficulty if the kidneys were unable to excrete it. Such a situation occurs not infrequently after severe operations, when renal function may be temporarily impaired and adrenal function accelerated. These patients lose water through the lungs and through other insensible channels, which requires replacement, but unless there has been vomiting or similar occurrence water alone is needed. In such cases the injection of much isotonic solution of sodium chloride has actually been shown to be deleterious.

A third condition in which isotonic solution of sodium chloride may provoke difficulty as compared with water without electrolyte is water deprivation in severe malnutrition with hypoproteinemia. In such a case the injection of isotonic solution of sodium chloride has been shown frequently to lead to so-called salt edema. While distilled water alone is rarely injected, 5 or 10 per cent glucose solutions without electrolyte is the usual method employed for the introduction of pure water. In this way an isotonic or slightly hypertonic solution enters the blood stream, after which the glucose is rapidly burned, leaving water itself.

These considerations, among others, emphasize the importance of distinguishing carefully between the need for water and the need for sodium chloride. The need for each or both must depend on the circumstances in the individual case. More important is the well established fact that excessive sodium chloride, unlike excessive water, is much more likely to prove deleterious and thus defeat the objectives of such parenteral injections.

ROBERT ELMAN, M.D., St. Louis.

CURARE THERAPY

To the Editor:—May I point out that the paper of Dr. Harold R. Griffith "Curare and Anesthesia," which was published in THE JOURNAL, March 17, is incomplete in its history of the use of the drug. He states that "clinicians had more than once cast hopeful eyes toward the possible use of curare in the treatment of spastic disease of the muscles but always its poisonous reputation and the presence of cardiac depressants and other adulterants in the available supplies made clinical trial seem too dangerous." This statement may have been true with specimens of curare antedating the use of the curare which was biologically assayed for me by Merck & Co. and used in many cases of spastic paralysis and allied conditions such as dystonia.

The first paper, "Curare Therapy for the Release of Muscle Spasm and Rigidity in Spastic Paralysis and Dystonia Musculorum Deformans," was published in the *Journal of Bone and Joint Surgery* in July 1938. A second study, "Therapeutic Use of Curare and Erythroidine Hydrochloride for Spastic and Dystonic States," was printed in the *Archives of Neurology* in February 1939. A third, "Clinical Experiences with Some Curare Preparations and Curare Substitutes," appeared in the *Journal of Pharmacology and Experimental Therapeutics* in June 1940, and, lastly, a summary of "Observations in Spastic Paralysis and Dystonia Musculorum Deformans" was read before the Boston Society of Psychiatry and Neurology on Oct. 20, 1938 and published in the *Journal of Nervous and Mental Disease* in June 1939.

These studies represent the first extensive use of curare with an assayed preparation.

I note also that the Council on Pharmacy and Chemistry has accepted the Squibb preparation of curare (THE JOURNAL, October 13). This preparation is an excellent one. I think it an error, however, to accept the trade name of the product. A similar policy, I believe, should be applied to all drugs to avoid confusing nomenclature.

MICHAEL BURMAN, M.D., New York.

USE OF GROUP O BLOOD IN TRANSFUSION

To the Editor:—In reply to a recent query concerning the use of A and B group substances to render group O blood safe for use for recipients of all blood groups, the statement was made that there is still no direct evidence establishing the efficacy of the group substances in vivo, when used in this way (THE JOURNAL, Nov. 17, 1945, p. 840). Our purpose in this communication is to describe briefly some recent experiments which supply this direct evidence.

Group B plasma having a very high titer of anti-A agglutinins was obtained from a blood donor who had been immunized by an injection of soluble group substances. The titer of the anti-A agglutinin for A_2 cells by the well slide method reached as high as 2,500 units (equivalent to 10,000 units by the tube technic or about two hundred times the average normal titer) but had fallen to a titer of 500 units by the slide technic by the time the plasma was collected from the donor some three months after the injection. A thousand cubic centimeters of this plasma was obtained at one time by withdrawing 1,800 cc. of blood from the donor and simultaneously replacing it with an equal volume of group B bank blood. As little as 25 cc. of the untreated plasma caused a frank hemolytic reaction in a voluntary group A_1 recipient, manifested by hemoglobinuria, pain in the back and increased bilirubinemia. Fifty cc. caused an even more pronounced reaction. In a volunteer of group A_2, 25 cc. of the plasma caused no obvious reaction but 50 cc. gave rise to signs of hemolysis. All these recipients were secretors.

To test the efficacy of Witebsky's A and B group substances to counteract the harmful effect of the plasma in question, Eli Lilly's preparation was added to the plasma in the proportion of 10 cc. for every 250 cc. of plasma. One group A_1 and one group A_2 volunteer was then each given 250 cc. doses of the neutralized plasma, and neither one exhibited any obvious manifestation of hemolysis. Similar results were obtained in a larger series of cases using other plasma. These results will be published shortly in detail, when the experiments have been completed.

LESLIE H. TISDALL, Major, M. C., A. U. S.
DONALD M. GARLAND, Captain, M. C., A. U. S.
ALEXANDER S. WIENER, M.D., Brooklyn.
LESTER J. UNGER, M.D., New York.

DETERMINATION OF RH FACTOR

To the Editor:—For almost a year I have been doing Rh factor tests on obstetric patients. The last six months I have been doing them routinely. The Nebraska State Health Department began offering this service about a month ago without charge. In my opinion it is a very definite advance in obstetrics.

Another great step forward will have been taken when all newborn babies, especially girls, have an Rh factor determination done at once as a part of the American newborn routine care. If the infant's test is Rh negative it should be rechecked in about three months because blood characteristics are not well developed in the first few days of life. I believe that a concerted effort ought to be put forth by the American Medical Association and other legitimate channels of propaganda, with the hope that the Rh factor determination will become a standard routine.

Those acquainted with the Rh factor realize that if this procedure is carried out and the Rh factor of each individual is obtained, the error of giving the wrong blood in infancy and from then on may be avoided. This once accomplished, there will be much less danger of the first baby having erythroblastosis. Also in case of emergency there will be a much better chance of obtaining the proper blood promptly.

RALPH LUIKART, M.D., Omaha.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Malpractice: Simultaneous Application of Tight Bandaging and Heat to Arthritic Foot.—Mrs. Treptau was suffering from what she thought was a sprain of her right foot and on Aug. 27, 1941 she went for treatment to the Behrens Spa, Inc., a hospital incorporated under the laws of Wisconsin and having as full time employees several chiropractors and at least one doctor of medicine. She was examined by Dixon, a chiropractor employed by the Spa, who advised her that she needed "vertebra adjustments," several of which were given to her by Dixon and by Behrens, another chiropractor, until September 14. In the course of those so-called treatments the swelling and pain in the foot increased greatly. September 14, a roentgenogram was taken under Behrens' direction at the Spa, and "by palpation in combination with the x-ray he determined that she was suffering from arthritis." He then applied a circular bandage consisting of two pieces of tape about 18 inches long and 1½ inches wide, starting one piece of tape about the top of the instep and going around the instep twice, and the other piece of tape went the other way. While applying the tape he told his patient that she had a bone infection and to grind her teeth as he tied the bandaging "real tight." This caused the patient a great deal of pain. Behrens then ordered "diathermo heat treatments" applied immediately. Two such treatments were given on each of the two following days. After the first heat treatment the patient was put in bed at the Spa. On her repeated complaints that the bandaging was too tight and that the pain was too great, a physician employee at the Spa apparently loosened the tape somewhat at the top of the foot but not around it. When the subsequent heat treatments were given the circular bandage was still in place and unloosened around the foot. The patient complained continually of great pain and the foot began to swell. On the 16th, the foot became black and blue, was very painful, and was so swollen that it covered the upper part of the adhesive tape bandage. On the failure of nurses attending her to bring a "doctor" to alleviate her pain, she induced the nurses to take the tape off her foot. The next day she returned to her home and called Dr. Wheelihan, a physician, in on the case. Dr. Wheelihan testified that when he first examined the patient he found the foot extremely swollen and very tender to touch, that this condition indicated a markedly impaired circulation, one of threatened gangrene which was the result of the impaired circulation. Just what the subsequent history of the case was the reported decision does not make clear except that at the time of the trial there was a loss of flexion and limitation in the foot, apparently a permanent condition.

The patient and her husband subsequently sued the defendant corporation, which operated the so-called Spa, for damages sustained by the patient as the result of alleged malpractice in the treatment rendered by the chiropractors employed by the Spa. On behalf of the plaintiffs three practicing physicians were called as witnesses, Dr. Wheelihan, who attended her after she left the Spa, and Drs. Werra and Nicely. Dr. Wheelihan testified that the probable and direct cause of the impaired circulation and the condition that he found when he first examined the patient was the constricting tape applied by chiropractor Behrens and the diathermic treatments ordered by Behrens while the tape was in place; that the treatment given was not in accord with the recognized proper practice of the community; that the combination of constricting bandage plus diathermy was not proper in any school or field of medicine; that a bandage constricts the circulation and heat tends to increase the circulation; that one or the other would have been proper, but not the combination; and that in his opinion such treatment and accentuation of the arthritic condition of the foot probably resulted in the present condition of the foot. The testimony of Drs. Werra and Nicely was to the same effect. The jury, in a special verdict, found that by the simultaneous bandaging and application of diathermic treatments the defendant failed to exercise the skill and care required by law and that failure was the cause of the disability suffered by the patient after treatment. The court accordingly entered judgment for the plaintiffs, and the defendant appealed to the Supreme Court of Wisconsin.

The defendant contended first that the trial court disregarded the well established principle of law that a physician is not liable for malpractice if he selects and uses one approved method of treatment although there may be testimony that if other expert witnesses had been treating the case they would have used another method or that the method employed was improper and that since there was testimony that the method employed by Behrens was a method of treatment recognized by the medical profession in that vicinity the defendant cannot be held guilty of malpractice. The trial court, however, said the court, consistently considered applicable the rule of law contended for by the defendant, as is exemplified in the following instruction it gave to the jury:

> You are further instructed that if you find from all the credible evidence in the case that the school of medicine recognizes more than one method of diagnosis or treatment of the condition which affected the plaintiff's right foot, it [defendant] was not required, at its peril, to select one or the other of such methods and was at liberty to select either of said methods and may not be considered wanting in the required degree of care and skill merely because expert witnesses give their opinion that some other method would have been preferable.

Consequently, the defendant's contention in this respect cannot be sustained.

The defendant next contended that a verdict in a malpractice case must be based on expert testimony that there was malpractice present and that the malpractice proximately caused the condition complained of. That rule of law, answered the court, is correct but again it is evident from the record that the trial court rightly concluded and duly instructed the jury that the plaintiffs must prove to a reasonable certainty by expert testimony that there was malpractice present and that such malpractice proximately caused such damages as the jury assessed. The jury was free to accept in determining those issues either the expert testimony offered by the physicians who testified for the plaintiff or the testimony of the chiropractors in the defendant's employ.

The defendant contended further that there was no credible evidence adduced at the trial to sustain to a reasonable certainty the jury's finding that the use of the treatments in combination by Behrens was the cause of the increased ailments, disabilities and suffering, or that such increase, if any, was a natural and

probable result of such use of treatments by Behrens. The defendant claimed that there was testimony that the patient's ailments, disabilities and pain could have been caused by the arthritic condition which affected her foot and that the burden of proof resting on the plaintiff is not met by showing that the ultimate condition might also have been caused by Behrens' unskilful treatment; that there was no testimony that the bandages were applied in such a manner as to impair circulation of the foot; that the claim that a tight bandage would aggravate an arthritic condition has no basis on medical fact; and that in the absence of gangrene no injury could result from the application of a tight bandage. This argument of the defendant, said the court, cannot be sustained. The jury's findings as to the defendant's malpractice and that it caused increased ailments, disabilities and suffering and damages is warranted by the testimony of the three physicians called by the plaintiffs.

The defendant next contended that medical practitioners such as were the three physicians called by the plaintiffs are not competent to testify as to the degree of care and skill required to be used by a chiropractor in treating a patient, relying on *Nelson* v. *Dahl*, 174 Minn. 574, 219 N. W. 941, reading in part as follows:

> When a patient selects one of the several recognized schools of treatment, he thereby adopts and accepts the kind of treatment common to that school; and the care, skill, and diligence with which he is treated, when that becomes a question in the courts of this state, must be tested by the evidence of those who are trained and skilled in that particular school of treatment.

The rule contended for, said the Supreme Court, is not applicable in view of the real issue in this case. Plaintiffs do not claim that there was malpractice on the part of the defendant while Behrens was engaged in the practice of chiropractic by chiropractic manipulations or adjustments of the spine. Instead, plaintiffs contend there was malpractice when he and his associates went beyond the practice of chiropractic and entered into the general field of the practice of medicine, by the application of the bandage to the foot and by giving in conjunction therewith the diathermo heat treatments, neither of which was the practice of chiropractic by chiropractic manipulation or adjustment of the spine. On the contrary, the application of the bandages and the heat treatments given in this case are part of the general practice of schools of medicine, and, in so far as there was thus an invasion of the general field of that practice, the methods thus used by defendant's employees in diagnosis and treatment were subject to the rules applicable in the practice of medicine and surgery. Consequently there was applicable in this case the rule that the considered opinion of a qualified member of the profession of medicine and surgery is competent and proper as to whether or not the treatment given constituted the required degree of care and skill which physicians in good standing in the community usually exercise. When there is such an invasion of the field of medicine by the treatment given, the rule, which confines the inquiry as to the required degree of a practitioner's skill and care to the rules and principles of chiropractic or the particular school of science or medicine to which he belongs, does not exclude the testimony of physicians of the other schools when that testimony bears on a point in relation to diagnosis or treatment as to which the principles of the schools do or should concur. When a chiropractor assumes to diagnose and to treat disease he must exercise the care and skill in so doing that is usually exercised by a recognized school of the medical profession. *Kuechler* v. *Volgmann*, 180 Wis. 238, 192 N. W. 1015.

The court also did not agree with a contention of the defendant that because it was a hospital and could not lawfully engage in the practice of medicine it could not be held liable for damages caused by malpractice in the performance of professional acts of its staff. In arriving at this conclusion the court quoted from 26 Am. Jur. p. 597, reading as follows:

> A hospital conducted for private gain is liable to its patient for injuries sustained by him in consequence of the incompetency or negligence of a physician treating him at its instance, under a contract binding it to furnish him proper treatment. A physician so employed is not an independent contractor.

The judgment in favor of the patient and her husband was accordingly affirmed.—*Treptau v. Behrens Spa, Inc., 20 N. W. (2d) 108 (Wis., 1945).*

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

NATIONAL BOARD OF MEDICAL EXAMINERS EXAMINING BOARDS IN SPECIALTIES

Examination of the National Board of Medical Examiners and the Examining Board in Specialties were published in THE JOURNAL, Dec. 22, page 1226.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: Mar. 19-21. Sec. Dr. B. F. Austin, 519 Dexter Ave., Montgomery 4.

ALASKA: Juneau, March 5. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

ARIZONA: * Phoenix, Jan. 4-5. Sec., Dr. J. H. Patterson, 826 Security Bldg., Phoenix.

CALIFORNIA: *Written.* Los Angeles, March 11-14. Sec., Dr. Frederick N. Scatena, 1020 N St., Sacramento 14.

CONNECTICUT: * *Examination.* New Haven, March 12-13. *Endorsement.* New Haven, March 26. Sec. to the Board, Dr. Creighton Barker, 258 Church St., New Haven.

DISTRICT OF COLUMBIA: * *Reciprocity.* Washington, March 11. Sec., Commission on Licensure, Dr. G. C. Ruhland, 6150 E. Municipal Bldg., Washington 1.

IDAHO: Boise, Jan. 15. Dir., Bureau of Occupational Licenses, Miss Agnes Barnhart, 355 State Capitol Bldg., Boise.

ILLINOIS: Chicago, Jan. 8-10. Supt. of Registration, Department of Registration & Education, Mr. Philip Harman, Springfield.

INDIANA: Indianapolis, April 25-27. Sec., Board of Medical Registration & Examination, Dr. W. C. Moore, 301 State House, Indianapolis 4.

KENTUCKY: Louisville, March 25-27. Sec., State Board of Health, Dr. Philip E. Blackerby, 620 S. Third St., Louisville 2.

MAINE: Portland, March 12-13. Sec., Board of Registration of Medicine, Dr. A. P. Leighton, 192 State St., Portland.

MICHIGAN: * Detroit, March 26-28. Sec., Board of Registration in Medicine. Dr J. E. McIntyre, 100 W. Allegan St., Lansing 8.

MONTANA: Helena, April 1-3. Sec., Dr. O. G. Klein, First National Bank Bldg., Helena.

NEVADA: Carson City, Feb. 4. Sec., Dr. G. H. Ross, 215 N. Carson St., Carson City.

NEW JERSEY: Trenton, June 18-19. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO: * Santa Fe, April 8-9. Sec., Dr. LeGrand Ward, 141 Palace Ave., Santa Fe.

NEW YORK: Jan. 28-31. Sec., Dr. Jacob L. Lochner, Education Bldg., Albany.

NORTH CAROLINA: *Reciprocity.* Raleigh, Jan. 16. Sec., Dr. Ivan Proctor, Raleigh.

NORTH DAKOTA: Grand Forks, Jan. 1. Sec., Dr. G. M. Williamson, 4½ S. Third St., Grand Forks.

OHIO: *Endorsement.* Columbus, Jan. 8. *Examination.* Columbus, March 19-22. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: * Oklahoma City, March 23. Sec., Dr. J. D. Osborn Jr., Frederick.

OREGON: * Jan. 23-26. Exec. Sec., Miss L. M. Conlee, 608 Failing Bldg., Portland 4.

PENNSYLVANIA: Harrisburg, January. Act. Sec., Bureau of Professional Licensing, Department of Public Instruction, Mrs. M. G. Steiner, 351 Education Bldg., Harrisburg.

RHODE ISLAND: * Providence, Jan. 3-4. Chief, Division of Examiners, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH DAKOTA: * Pierre, Jan. 15-16. Sec., Medical Licensure, State Board of Health, Dr. Gilbert Cottam, Capitol Bldg., Pierre.

TEXAS: Austin, March 27-29. Sec., Dr. T. J. Crowe, 918-20 Texas Bank Bldg., Dallas 2.

VERMONT: Burlington, March or April 1946. Sec., Dr. F. J. Lawliss, Richford.

WASHINGTON: * Seattle, Jan. 14-16. Dir., Department of Licenses, Mr. Harry C. Huse, Olympia.

WEST VIRGINIA: Charleston, Jan. 7-9. Commissioner, Public Health Council, Dr John E. Offner, State Capitol, Charleston 5.

WISCONSIN: * Madison, Jan. 8-10. Sec., Dr. C. A. Dawson, Tremont Bldg., River Falls.

WYOMING: Cheyenne, Feb. 4. Sec., Dr. G. M. Anderson, Capitol Bldg., Cheyenne.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

CONNECTICUT: Feb. 9. Address State Board of Healing Arts, 250 Church St., New Haven 10.

FLORIDA: Gainesville, June 4. Final date for filing application is May 20. Sec. Dr. J. F. Conn, John B. Stetson University, DeLand.

IOWA: Des Moines, Jan. 8. Dir., Division of Licensure & Registration, Mr. H. W. Grefe, Capitol Bldg., Des Moines.

MICHIGAN: Ann Arbor and Detroit, Jan. 11-12. Sec., Miss Eloise LeBeau, 101 N. Walnut St., Lansing.

MINNESOTA: Minneapolis, Jan. 2-3. Sec., Dr. J. C. McKinley, 126 Millard Hall, University of Minnesota, Minneapolis 14.

NEBRASKA: Omaha, Jan. 8-9. Dir., Bureau of Examining Boards, Mr. Oscar F. Humble, 1009 State Capitol Bldg., Lincoln 9.

OKLAHOMA: Oklahoma City, Jan. 21. Sec., Dr. J. D. Osborn Jr., Frederick.

OREGON: Portland, March 2. Sec., Board of Higher Education, Mr. C. D. Byrne, University of Oregon, Eugene.

RHODE ISLAND: Providence, Feb. 13. Chief, Division of Examiners, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH DAKOTA: December. Sec., Dr. J. D. Alway, Aberdeen.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1935 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

Alabama State Medical Assn. Journal, Montgomery

15:89-112 (Sept.) 1945

Use of Aluminum in Silicosis Control. I. R. Tabershaw and B. D. Tebbens.—p. 89.
Erythroblastosis Fetalis, or Hemolytic Disease of the Newborn. M. V. Adams.—p. 93.
Lymphosarcoma of Appendix: Report of Case. W. E. Wilson.—p. 98.
Hospitals Now and Tomorrow. A. C. Bachmeyer.—p. 100.

15:113-148 (Oct.) 1945

Alabama Doctor. W. H. Brantley Jr.—p. 113.
Prepayment Medical Care for State of Alabama. A. C. Jackson.—p. 126.
Marion Sims and Other 19th Century Pioneers: Dawn of Scientific Medicine and Surgery. S. Harris.—p. 128.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

54:217-328 (Sept.) 1945

Electrokymograph for Recording Heart Motion Utilizing Roentgenoscope. G. C. Henny and B. R. Boone.—p. 217.
Entrance of Pantopaque into Venous System During Myelography. C. L. Hinkel.—p. 230.
Osteogenic Sarcoma and Chondrosarcoma, with Special Reference to Roentgen Diagnosis. E. P. Pendergrass, J. O. Lafferty and R. C. Horn.—p. 234.
*Teratoma of Testis: Report of 65 Cases. J. L. Barner.—p. 257.
Atypical Esophageal Displacement with Left Atrial Dilatation, with Notes on Rheumatic Aortitis. B. S. Epstein.—p. 262.
Anomalous Right Subclavian Artery. B. Copleman.—p. 270.
Roentgenologic Diagnosis of Benign Tumors (Single Polyps) of Colon. F. J. Lust.—p. 276.
*Roentgen Irradiation in Treatment of Marie-Strümpell Disease (Ankylosing Spondylarthritis): Analysis of 160 Cases. J. E. Hemphill and R. J. Reeves.—p. 282.
Influence of Biologic Factors on Form of Roentgen Ray Survival Curves: Experiments on Paramecium Caudatum. A. Back and L. Halberstaedter.—p. 290.
Calculation of Radium Dosage Along Longitudinal Axis of Linear Sources. B. S. Wolfe.—p. 296.

Teratoma of Testis.—Barner reports 65 cases of teratoma of the testis treated within thirty-three months at an army general hospital. This was 2.62 per cent of all cases admitted to the genitourinary service and 7.86 per cent of all cases of cancer. The following factors have been suggested as contributory to the etiology of these malignant tumors: heredity, abnormal development, sexual activity, trauma, occupation, biochemical or endocrine changes and infections. Of these the most significant is probably abnormal development. The growth of this tumor is usually insidious and the patient may not be aware of it for some time. Painless swelling is the most frequent symptom. The testis usually retains its natural shape and outline. Early diagnosis is the most important factor in a high survival rate. Treatment should rely on surgery and postoperative irradiation. Orchiectomy and removal of the cord with the accompanying structures high at the internal abdominal ring are indicated in every case because it makes possible the removal of much of the lymphatic tract and contributes to the postoperative comfort of the patient. A remaining stub of the cord is not only a potential site for recurrence but is also tender and a source of irritation and discomfort. External high voltage roentgen therapy is begun as early as the third or fifth day after operation with 220 kilovolts, 15 milliamperes, 50 cm. target-skin distance, 0.5 to 1 mm. of copper plus 1 mm. of aluminum as filter and 47 to 32 roentgens per minute (measured in air). The number of fields irradiated depends on the presence or absence of metastases and the duration of symptoms before operation. Irradiation is given daily except Sundays. The daily amounts of radiation are 200 to 250 roentgens to each of two fields, in rotation, until a total of 1,600 to 2,000 roentgens per field has been administered. Follow-up revealed that 7 of the 65 patients have died.

Roentgen Irradiation in Ankylosing Spondylarthritis.—Hemphill and Reeves present an analysis of 160 cases of Marie-Strümpell disease in which the pathologic picture is a true atrophic arthritis. The course is that of a progressive deforming arthritis unless correction and prevention of the deformities are instituted. During the earliest inflammatory stage of the disease roentgenograms may be negative. Forty-five degree angle roentgenograms may show changes about the articular facets of the vertebrae. The facets of the twelfth dorsal vertebra are frequently affected earlier than others. Usually the earliest detected changes are in the sacroiliac joints and consist of decalcification and irregular trabeculation in the subchondral bone immediately adjacent to the joint space, with haziness and apparent widening of the joint space. Of the 160 patients 137, or 86 per cent, were male. More than three fourths of the patients were between 20 and 39 years of age. Muscle spasm may exist for years before ossification of ligaments takes place. Roentgen therapy finds its greatest usefulness in allowing orthopedic treatment and correction of the deformities to be tolerated by the patient. During roentgen treatment, which employs 200 kilovolts at 30 to 50 cm. distance with a filter of 0.5 mm. of copper plus 1 mm. of aluminum, the spinal column is blocked off by lead shields in such a way that the lateral spinal ligaments are included in the beam. Two or three fields are treated every day or every other day with 150 roentgens in air to each field until a total of 450 or 600 roentgens has been applied per field. The series of high voltage roentgen treatments can be repeated at the end of six or eight weeks. The pathologic granulations of Marie-Strümpell disease are similar to the vascular granulations of other inflammatory reactions known to be radiosensitive. Whether or not roentgen therapy arrests this granulation before ankylosis develops cannot be answered. It is felt, however, that if roentgen therapy can be instituted while there is still active granulation (and not complete ankylosis) good results may be expected.

American Journal of Surgery, New York

70:1-136 (Oct.) 1945

Established Surgical Infections: Treatment with Urea Sulfanilamide Mixture. C. W. Brown, L. A. McClintock and E. R. Neary.—p. 4.
Ligation of Supernumerary Ureter: Clinical and Experimental Study. A. E. Goldstein and B. Klotz.—p. 13.
Intervertebral Spine Fusion with Removal of Herniated Intervertebral Disk. J. M. Ovens and H. G. Williams.—p. 24.
Fracture of Zygomatic Bone and Arch Postoperative Headgear. A. A. Schmier.—p. 27.
Acute Suppurative and Gangrenous Cholecystitis. N. Blumberg and L. Zisserman.—p. 38.
Metatarsal March (Fatigue) Fractures. A. L. Leveton.—p. 49.
Chlorophyll and Adrenal Cortical Extract in Local Treatment of Burns. G. H. Collings Jr.—p. 58.
Esophageal Diverticulum. F. Bortone.—p. 64.
Modifications of Trocar Method with Positive Pressure for Instilling Amniotic Fluid Concentrate Intra-Abdominally at Close of Operations. H. J. Merkle.—p. 68.
Modification of Radical Operation for Cure of Ingrown Toenails. W. L. Sibley.—p. 79.
What Was Wrong with Whitehead's Work? Appraisal of His Rectal Operation. C. Eaton.—p. 83.

Archives of Physical Medicine, Chicago

26:549-602 (Sept.) 1945

Dr. Simon Baruch and His Fight for Free Public Baths. T. E. Keys and F. H. Krusen.—p. 549.
Physiologic Fundamentals of Spa Therapy. J. Goldberger.—p. 558.
Spa Treatment of Rheumatic Diseases in United States. R. Kovacs.—p. 567.
*Challenge of Crutches: III. Standard Crutch Gaits and How to Teach Them. G. G. Deaver and Mary E. Brown.—p. 573.
Artificial Fever Chemotherapy: V. Induction Period. R. M. Craig, F. M. Buroker and G. X. Schwemlein.—p. 582.
Responsibility of Physical Therapist to Community. D. W. Gudakunst.—p. 590.

Crutch Gaits.—Deaver and Brown think that all crutch walkers should learn at least two gaits: a fast one to be used in the open, or in crossing a street, and a slow one for crowded places where space is limited but balance must be kept as slow progression is made. A patient should be taught as many

crutch gaits as he can master. He should know more than one, because each crutch gait requires a different combination of muscles, and a disabled person who becomes fatigued with one may change to another. Each crutch gait can be used as an exercise. The most elementary standard crutch gait consists in advancing one crutch, then the opposite foot, then the other crutch and finally the other foot. This four point alternate gait is a safe gait because there are always three points of support on the floor. It is a slow gait because weight must be constantly shifted. It does not require much space, so that it is a good gait to use in crowds or where space is limited. Once the four point alternate gait has been mastered, the subject may be taught next the two point alternate, which involves placing one crutch and the opposite foot down on the floor simultaneously, then the other crutch and the other foot simultaneously. This is no more than a speeding up of the four point alternate gait. This is a faster gait than the four point. The three point crutch gait is the one in which the two crutches and the weaker limb are placed on the floor simultaneously, then the stronger limb, which, being strong enough to support the weight of the body, needs no crutches. This gait is used by those having one lower extremity which cannot take full weight bearing and one which can support the whole body weight. The author describes the tripod crutch gaits and the swinging crutch gaits, also the rocking chair crutch gait and the amputation crutch gaits. The four point alternate and the two point alternate crutch gaits are taught to amputation subjects. The subject with artificial limbs is supported by his artificial limbs. He does not need the crutches for support. He needs them for balance. The aim is to use crutches at first for balancing purposes until the subject has "the feel" of his artificial limbs.

Endocrinology, Springfield, Ill.

37:157-222 (Sept.) 1945

Plasma Protein Concentrations and Organ Weights of Rats on High Protein Diet. J. H. Leathem.—p. 157.
Effectiveness of Gonadotropin Injections Followed by Insemination in Inducing Pregnancy in Ewes. M. Koger.—p. 165.
Study of Hyaluronidase Effects on Follicle Cells of Ovulated Rat Ova. S. L. Leonard and R. Kurzrok.—p. 171.
Ultrafiltration of Urine for Collection and Biologic Assay of Excreted Hypophysial Hormones. A. Gorbman.—p. 177.
*Effects of Pancreatectomy on Glycosuria and Ketosis in Dogs Made Diabetic by Alloxan. Elizabeth Thorogood and B. Zimmermann. —p. 191.
Effects of Alloxan in Birds. C. C. Scott, P. N. Harris and K. K. Chen. —p. 201.
Effect of Alcohol and Potassium Hydroxide Concentration on Reaction of 17-Ketosteroids with M-Dinitrobenzene. Hildegard Wilson and I. T. Nathanson.—p. 208.

Pancreatectomy and Alloxan Diabetic Dogs.—Thorogood and Zimmermann observed that, although glycosuria is more severe and the insulin requirement higher in alloxan treated than in pancreatectomized dogs, the former are able to live much longer without insulin treatment and fail to develop ketosis and diabetic coma. This has been confirmed by pancreatectomy in alloxan diabetic dogs following which the insulin requirement is less, but the animals pass quickly into coma when not controlled with insulin. One hypothesis suggests that there is secreted by the pancreas a second endocrine factor which acts to increase the blood sugar but to prevent ketosis in the insulin deficient animal. This hypothesis is a tentative one which seems to be the simplest explanation of the data, though it can be confirmed only by the actual preparation of an extract possessing these types of activity.

Florida Medical Association Journal, Jacksonville

32:107-166 (Sept.) 1945

Acute Rheumatic Fever. J. E. Shay.—p. 135.
Modification of Tyding and Tivnen Tonsil-Seizing Forceps. R. R. Killinger.—p. 137.
History of Medicine in Duval County: Part VI. W. Merritt.—p. 137.

32:167-214 (Oct.) 1945

Successful New Method of Suspending Prolapsed Transverse Colon. M. Smith.—p. 191.
Benzedrine Sulfate Therapy for Urticaria in Children. S. J. Roberts. —p. 193.
Medicine in This Changing World. J. B. Quicksall.—p. 194.

Hawaii Medical Journal, Honolulu

5:7-40 (Sept.-Oct.) 1945

Uterine Inertia and Postpartum Hemorrhage. L. T. Heywood.—p. 9.
*Spontaneous Complete Rupture of Normal Uterus During Late Pregnancy Before Onset of Labor. G. C. Milnor.—p. 12.
Continuous Caudal Anesthesia in Obstetrics. J. Herzlich.—p. 15.
Endocrine Therapy in Functional Uterine Bleeding. A. M. Faris.—p. 17.
Management of Breech Delivery. H. E. Bowles.—p. 19.
Present Status of Organotherapy in Essential Dysmenorrhea. E. R. Muntz.—p. 21.
Mammaplasty of Pendulous Breasts. C. E. Fronk.—p. 23.
Management of Occipitoposterior Positions. A. M. Faris.—p. 26.

Spontaneous Rupture of Uterus During Late Pregnancy.—Milnor reports the case of a primigravida aged 27 whose pregnancy had progressed normally up to three weeks before the expected date of delivery on March 22, 1945. At 3 a. m. on March 2 she was awakened from a sound sleep by the urge to urinate. After voiding she went back to bed. One-half hour later a most severe abdominal pain started with nausea followed by several very profuse and loose stools. She had retired at 9:30 p. m. feeling fine and had led a normal life the day before. The distress grew worse. She felt "as though something had burst" within her. Breathing became difficult and great substernal distress was noticed. She felt faint. She did not arrive at the hospital until three hours after the onset of the pain. When she arrived, after a 10 mile taxi ride, she was in extreme shock. She continued to vomit but said that the pains were a little less severe. The abdomen was tender and distended. Its contour was very irregular, saddle-like, with a mass in the upper left quadrant and another in the suprapubic region. A diagnosis of ruptured uterus was made. One-half hour elapsed between the time of diagnosis and laparotomy, four hours after the first pain. A 7 pound (3,175 Gm.) stillborn child was found free in the upper left abdomen. The placenta was found to be plugging a 6 cm. rent in the right posterior wall of the uterus. Blood clots and amniotic fluid were present in large amounts in the abdominal cavity. The child was quickly delivered and the placenta extracted. The uterus was removed subtotally. The patient recovered rapidly. The cause of this sudden accident three weeks before term, and before the onset of labor, is unexplained. There was no disease of the uterus demonstrated. There may have been a sudden violent movement of the fetus, which in turn caused a violent contraction lacerating the uterus.

Journal-Lancet, Minneapolis

65:265-314 (Aug.) 1945

Kenny Concept and Treatment of Infantile Paralysis: Report of Five Year Study of Cases Treated and Supervised by Miss Elizabeth Kenny in America. J. F. Pohl.—p. 265.
Observations on Tropical Disease. J. A. Holmes.—p. 272.

65:315-350 (Sept.) 1945

Body Minerals. W. G. Richards.—p. 315.
*Tuberculosis Among College Students: Fourteenth Annual Report of Tuberculosis Committee, American Student Health Association, for Academic Year 1943-1944.—p. 322.

Tuberculosis Among College Students.—The fourteenth annual report of the Tuberculosis Committee of the American Student Health Association covers the reported case finding programs in 286 colleges during 1943-1944. Of 282 colleges, 199 include a tuberculin testing routine and 83 employ chest roentgenograms without preliminary testing. Student enrolment at institutions having survey programs was 286,018. Among these 622 new cases of tuberculosis were diagnosed, a rate of 217.4 per hundred thousand. The lesions were classified as unstable in 156 and quiescent in 151; 315 were designated as healed. Students withdrawing from college to undergo treatment numbered 169. The committee emphasizes the need for active treatment in the majority of cases of tuberculosis found among young men and women of college age. Of the 622 students discovered to have early tuberculosis 453 were permitted to remain in college. It seems highly probable that a considerable proportion of this group may experience progression of their disease and will eventually have to undergo treatment. The two dose Mantoux method is recommended as the method of choice in tuberculin testing. If a single test dose is employed, an intermediate dose of at least 0.1 mg. old tuberculin or 0.0001 mg. of purified protein derivative should be used. The

Vollmer patch test cannot be recommended for use in colleges. It is sound practice and in the interests of economy to provide chest roentgenograms for only those students who react to an adequate dose of tuberculin. Complete protection against tuberculosis for college students cannot be attained through a program limited to the student body. Faculty members and employees, including food handlers, should participate in the tuberculosis control program. The lesions of pulmonary tuberculosis encountered in college students are, in a majority of instances, unstable and potentially dangerous. The absence of symptoms does not preclude the necessity for early treatment. Students with pulmonary lesions who remain in college should be under close observation, with frequent clinical and roentgenographic studies.

Maine Medical Association Journal, Portland

36:149-166 (Sept.) 1945

Presidential Address. R. V. N. Bliss.—p. 149.
New Wagner-Murray-Dingell Bill and S. 191. G. Bugbee.—p. 151.
Hospitals Now . . . and Tomorrow. A. C. Bachmeyer.—p. 156.

36:167-182 (Oct.) 1945

Physicians as Metaphysicians. J. S. Bixler.—p. 167.
Intraspinal Myelography and Herniated Intervertebral Disk in Lumbar Spine. R. R. Rix.—p. 169.

New England Journal of Medicine, Boston

233:339-368 (Sept. 20) 1945

Diagnosis and Treatment of Cystitis in Women and Children. H. L. Kretschmer.—p. 339.
Glycosuria and Hyperglycemia Associated with Acute Meningitis: Report of Case. J. J. Federer.—p. 342.
One-Piece Munro Tidal Drainage Apparatus. W. A. R. Chapin.—p. 344.
Physiology. H. E. Hoff.—p. 345.
Acute Rheumatic Fever. Severe Rheumatic Myocarditis.—p. 359.
Portal Cirrhosis of Liver, Alcoholic Type.—p. 362.

233:369-398 (Sept. 27) 1945

Hodgkin's Disease: V. Involvement of Certain Other Organs. H. Jackson and F. Parker Jr.—p. 369.
Effect of Prediabetic State on Survival of Fetus and Birth Weight of Newborn Infant. H. C. Miller.—p. 376.
Parenteral Use of Vitamin Preparations. F. J. Ingelfinger.—p. 379.
Dissecting Aortic Aneurysm, with Involvement of Coronary Mouths, Occlusion of Left Femoral Artery and Rupture into Pericardium. —p. 386.
Nephrosis, Probably Due to Bismuth Intoxication. Syphilitic Aortitis. —p 389.

233:399-426 (Oct. 4) 1945

Acute Pericarditis: Graphic Correlation of X-Ray, Clinical and Electrocardiographic Findings. D. L. Urschel, P. K. Bondy and S. M. Salley.—p. 399.
*False Positive Hinton Reactions Following Chickenpox. L. W. Kane and P. H. Henneman.—p. 407.
Parenteral Use of Vitamin Preparations. F. J. Ingelfinger.—p. 409.
Cerebral Infarct, Right: Internal Capsule and Corpus Striatum. Carcinoma of Stomach, with Extension to Omentum and Metastases to Liver and Mediastinal and Retroperitoneal Lymph Nodes.—p. 418.
Tubal Pregnancy.—p. 420.

Hinton Reactions Following Chickenpox.—Kane and Henneman during the last eighteen months observed the occurrence of transient false positive Hinton reactions in patients convalescent from chickenpox. The cases of chickenpox include 11 occurring in Harvard University students and 11 in children. All bloods were carefully drawn and the tests were carried out by the same skilled technician using undiluted serum. Wassermann, Hinton and Kahn tests were performed on each specimen. Five of the 22 patients with chickenpox exhibited transient false positive Hinton reactions for syphilis. In addition, 1 patient developed a transient positive Kahn reaction. No false positive Wassermann reactions were obtained.

New Jersey Medical Society Journal, Trenton

42:283-312 (Sept.) 1945

Primary Atypical Pneumonia: Statistical Analysis. W. A. Tansey, P. J. Culver and T. F. Frist.—p. 288.
Appendicitis. F. W. Rice.—p. 294.
Shock Therapy: 1937 to 1944. J. H. Taylor.—p. 300.

42:313-346 (Oct.) 1945

Fractures of Head of Radius. B. L. Clement.—p. 317.
Local Reaction to Penicillin Given by Mouth. E. C. Kern.—p. 326.
Board of Children's Guardians Health Program. J. E. Alloway.—p. 326.
Radical Resection of Carcinoma of Rectum and/or Rectosigmoid in Continuity with Retention of Anal Sphincter. A. O. Wilensky.—p. 328.

Pennsylvania Medical Journal, Harrisburg

48:1217-1408 (Sept.) 1945

Report on Poliomyelitis in Allegheny County, 1944. W. H. Robinson and W. R. Sweadner.—p. 1233.
Observations on Acute Poliomyelitis: Outbreak of 1944 in Allegheny County, Pa. J. Wright.—p. 1238.
Lobectomy and Pneumonectomy in Modern Medicine. C. P. Bailey. —p. 1242.
Recent Concepts of Endocrine Therapy in Women. R. H. Friday. —p. 1245.

49:1-96 (Oct.) 1945

Carcinoma of Colon in Rural Pennsylvania. H. L. Foss.—p. 17.
Fusospirochetal Pulmonary Abscess: Clinical Report of Case. E. R. Wiese and C. A. Heiken.—p. 28.
Movement for Improvement in Health and Physical Fitness: What Role Should We Play in Pennsylvania? L. G. Rowntree.—p. 31.

Public Health Reports, Washington, D. C.

60:1129-1164 (Sept. 28) 1945

*Chemotherapeutic Action of Streptomycin and Promin in Experimental Tuberculosis. M. I. Smith and W. T. McClosky.—p. 1129.
Outbreak of Food Poisoning Due to New Etiologic Agent: Salmonella Berta. G. H. Hauser, W. L. Treuting and L. A. Breiffelh.—p. 1138.

60:1165-1200 (Oct. 5) 1945

Therapeutic Effect of Promin in Leprosy. G. H. Faget and R. C. Pogge. —p. 1165.
Sickness Absenteeism Among Industrial Workers, Second Quarter of 1945, with Inquiry into Occurrence of Digestive Diseases, 1936-1945. W. M. Gafafer.—p. 1179.

Streptomycin and Promin in Experimental Tuberculosis.—Smith and McClosky report that daily intramuscular injections of 5,000 units of streptomycin for a period of ninety days in guinea pigs infected with a human strain of tubercle bacilli has produced a chemotherapeutic effect superior to that obtained with 0.5 Gm. of promin per kilogram of weight given orally for the same length of time. Since the dose of promin used is about half the maximum tolerated dose while the dose of streptomycin is less than one-twentieth the toxic dose, it appears that streptomycin has a chemotherapeutic index better than ten times that of promin. It also seems possible that by increasing the dose of streptomycin and with better methods of administration its chemotherapeutic effectiveness may be enhanced. Using a suitable combination of streptomycin and promin, it was possible to obtain better results under the experimental conditions employed by the authors than have ever been obtained previously.

Quarterly J. of Studies on Alcohol, New Haven, Conn.

6:131-270 (Sept.) 1945. **Partial Index**

Simple Method for Determination of Alcohol in Micro Quantities of Blood and Other Fluids. L. A. Greenberg.—p. 135.
New Legislation for Control of Alcoholism: Connecticut Law of 1945. S. D. Bacon.—p. 188.
Attitude of Industrial Management Toward Alcoholism. C. Mortenson. —p. 205.
Screening Inebriates in Municipal Courts. L. Drucker.—p. 209.
Physician and Alcoholic. H. W. Haggard.—p. 213.
Role of Church and Pastor in Postwar Alcoholism. O. F. Blackwelder. —p. 222.
Alcoholic in Penal Institution. H. B. Gill.—p. 233.
Alcoholics Anonymous in Postwar Emergency. W. W., One of the Founders.—p. 239.
Note on Drinking in College Community. C. C. Fry.—p. 243.
The Citizen's Part in Problem of Alcoholism. M. Mann.—p. 249.

Review of Gastroenterology, New York

12:321-392 (Sept.-Oct.) 1945

Gastric Acidity and Occult Blood Studies of Young Adult Males with Duodenal Ulcer. R. C. Page.—p. 343.
Gastrostomy: History of Its Development, Indications and Contraindications. M. Thorek.—p. 347.
Pelvic and Abdominal Pain in Women. C. A. Gordon.—p. 353.
Abdominal Pain in Functional Disorders. W. Ostrow.—p. 356.
Testicle and Gastric Secretion. P. DeMuro and F. Marconi.—p. 363.

Rhode Island Medical Journal, Providence

28:625-700 (Sept.) 1945

Newer Developments in Etiology and Treatment of Diabetes. E. P. Joslin.—p. 639.
Critic of Psychosomatic Medicine. C. Binger.—p. 642.
Geriatrics. R. I. Lee.—p. 647.
The Doctor's Patient and Wagner Act. J. F. Kenney.—p. 652.
Industrial Accidents: Comparative Study of Accident Record at Collyer Insulated Wire Company, Pawtucket, R. I. C. L. Farrell and H. Stanzler.—p. 661.

Southern Medical Journal, Birmingham, Ala.

38:565-634 (Sept.) 1945

Rectal Ulcers with Perirectal Fistula as Port of Entrance of Torula Encephalitis. V. K. Allen and L. Lowbeer.—p. 565.
Modified Manchester Technic for Partial Prolapse of Uterus. L. F. Turlington.—p. 570.
Carotid Sinus Syndrome. W. K. Purks.—p. 578.
Echinococcus Disease: Report of 3 Cases of Calcified Cysts of Liver. P. L. Hudson.—p. 584.
Note on Influence of "Folic Acid" on Leukocyte Equilibrium in Malnourished Patients. L. J. Berry, T. D. Spies and C. A. Doan.—p. 590.
Contagiousness of Poliomyelitis: Studies in Bacterial Metabolism: CXXI. A. I. Kendall.—p. 593.
*Role of Adrenal in Waterhouse-Friderichsen Syndrome: Case Report. A. H. Loudon Jr. and R. Holman.—p. 596.
Treatment of Asphyxia. J. Kreiselman.—p. 598.
*Results of Surgical Treatment in 100 Cases of Chronic Mental Illness. L. Hofstatter, A. K. Busch, J. F. Clancy and E. A. Smolik.—p. 604.
Kidney Colic Simulated by Traumatic Dissecting Aneurysm of Abdominal Aorta. H. E. Carlson.—p. 607.
Some Experience with Small Dosage Dust and Pollen Therapy. F. K. Hansel.—p. 608.
Relationship of Radiologist to Practicing Physician. R. T. Wilson. —p. 613.
Undergraduate Curriculum in Medicine. J. P. Gray.—p. 619.
Medical Education Above Undergraduate Level. H. W. Kostmayer. —p. 624.

The Adrenal in the Waterhouse-Friderichsen Syndrome.—London and Holman report 5 cases of Waterhouse-Friderichsen syndrome. One patient had meningococcic meningitis and appeared moribund on admission but recovered following intensive sulfadiazine therapy. Four of the patients were diagnosed clinically as having Waterhouse-Friderichsen syndrome. Only 3 of these patients presented postmortem massive adrenal hemorrhage necessary to fulfil the criteria for the diagnosis of this syndrome. There was nothing in the clinical or laboratory data to distinguish the case that failed to meet the criteria from those which did. One case is unique in that the typical symptoms with positive blood cultures (meningococcus type II-A) and massive adrenal hemorrhage occurred in a woman aged 30 who was eight months pregnant. The fetal blood culture was positive for the same organism, but the fetal adrenals appeared normal. This case provides the human counterpart of the experiments of Stewart and Rogoff, who showed that bilateral adrenalectomy was without effect in pregnant dogs because ample cortical hormone was supplied by the fetal adrenals. The authors believe that the currently accepted view that death in cases of Waterhouse-Friderichsen syndrome is due to adrenal insufficiency secondary to hemorrhage in the adrenal glands is untenable and that the indications for adrenal cortex therapy are as great in all cases of severe septicemia and toxemia as they are in cases of Waterhouse-Friderichsen syndrome.

Surgical Treatment of Chronic Mental Illness.—Hofstatter and his associates review the clinical results obtained with bilateral prefrontal leukotomy in the first hundred institutionalized patients operated on since 1941. This series represents patients who have been ill for an average of ten years and have been hospitalized for an average of five years. Many of these showed not only social maladjustment but definite antisocial behavior on a noisy, combative and destructive level, often with suicidal or homicidal tendencies. They had received many other types of therapy, all of which had failed to influence the psychosis. In 65 of the patients all four quadrants were cut. In 35 patients leukotomy was limited to the lower (orbital) quadrants exclusively, with no statistical difference in therapeutic effect. Comparing the results in these 100 patients with those obtained in a survey of 582 patients in seventeen other centers, the authors find that the percentages lie close together; they report 33 per cent much improved, 34 per cent improved and 31 per cent slightly or not improved, which compares favorably with 31, 33 and 30 per cent. They think that the results justify the use of this procedure in the chronic mentally ill.

Virginia Medical Monthly, Richmond

72:403-454 (Oct.) 1945

Influence of Sister Kenny Publicity on Treatment of Poliomyelitis. R. V. Funsten.—p. 403.
Brain Tumor in State Hospital Patients: Study of 8 Cases in 120 Consecutive Autopsies. E. L. Crumpacker and W. Riese.—p. 407.
Somatic Neuroses. F. H. Redwood.—p. 420.
Connecting Link. J. H. Hagy.—p. 425.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Medical Journal, London

2:309-342 (Sept. 8) 1945

*Meningitis After Spinal Analgesia. M. Kremer.—p. 309.
Treatment of Burns: Plea for Simplicity. C. W. Flemming.—p. 314.
First Aid Treatment of Phosphorus Burns. W. McCartan and E. Fecitt. —p 316.
Globin Insulin: Clinical Trial. J. M. Malins.—p. 318.
Case of Paraplegia After Cerebrospinal Meningitis. H. H. Stewart. —p. 319.
Fractures of Internal Malleolus. G. M. Muller.—p. 320.

2:343-376 (Sept. 15) 1945

Thiouracil in Treatment of Thyrotoxicosis. A. Grainger, D. A. Gregson and H. S. Pemberton.—p. 343.
Role of Animal Type Dermatophytes in Human Ringworm. B. A. Thomas, Mary Lennox and J. T. Duncan.—p. 346.
Nerve Injuries in Children. H. S. Souttar.—p. 349.
Sciatic "Neuritis." J. M. Holmes and B. R. Sworn.—p. 350.
Food Consumption of Working Class and Lower Middle Class Families in Brussels During January 1945. P. A. Bastenie, T. F. Macrae, G. A. Smart and S. Yudkin.—p. 351.
Case of Local Tetanus. F. A. Elliott.—p. 353.

Meningitis After Spinal Analgesia.—Kremer reports 7 cases of infection of the subarachnoid space after spinal analgesia. In 1 the necropsy showed a chronic inflammation of the subarachnoid space, which had resulted in fibrin deposition with subsequent organization. In this process of organization pools of infected cerebrospinal fluid had become isolated. If the fibrin should break down, this infected cerebrospinal fluid would be liberated into the general subarachnoid circulation. These isolated areas may not develop the same concentration of therapeutic substances as is present in the free flowing cerebrospinal fluid. If the causative organism is sensitive to sulfonamides it is cleared only from the general cerebrospinal fluid circulation and not from these loculi. These areas of isolated cerebrospinal fluid occasionally are freed and cause the relapses which form the most striking clinical feature of the infection. There can be little doubt that the spinal analgesics are mild irritants; it is possible that this irritation results in the outpouring of protein rich fluid, which provides a favorable medium for pathogenic organisms. The only satisfactory treatment is prophylaxis. It should be possible to avoid these cases. If in an emergency a rigid technic is not possible, spinal analgesics should not be used.

Edinburgh Medical Journal

52:289-336 (Sept.) 1945

Physiology of Peripheral Nerve Injury. A. E. Ritchie.—p. 289.
Shock Due to Tissue Trauma: Observations on Diagnosis and Assessment. A. W. Wilkinson.—p. 306.
Problem of Stiff Knee Joint in Fracture of Shaft of Femur. I. S. Smillie.—p. 317.
Severe Hemorrhage from Ascending Colon Treated by Ligation of Ileocolic Artery: Report of Case. D. L. C. Bingham.—p. 329.

Journal of Royal Naval Medical Service, London

31:129-194 (July) 1945

Salt Deficiency States in Tropical Climates. J. C. Stenning.—p. 129.
*Working Conditions of Boiler Cleaning Parties with Special Reference to Dust Inhalation. G. D. Channell.—p. 146.
Gunnery and Blast Deafness. S. C. Suggitt.—p. 151.
Bubonic Plague in Dakar. C. M. H. Rotman.—p. 155.
Medical Study of the Black Hole of Calcutta. M. Critchley.—p. 158.
Pyoderma in the Tropics, with Description of 3 Cases. A. R. Harper. —p. 170.

Boiler Cleaning and Dust Inhalation.—The investigation carried out by Channell showed that dust is produced in large quantities during the cleaning of boilers. Dust masks are essential to protect boiler cleaners. The type obtained for trial has proved very satisfactory. A single analysis of boiler tube deposit showed that it contained a high percentage of metallic copper and small amounts of metallic oxides, notably lead. If inhaled, these metals are detrimental to health. Further analyses of boiler deposit are required to confirm the constant presence of these constituents. Protective clothing would increase the comfort of working conditions. Eye shields are admirable for the protection of the eyes. The incidence of upper respiratory infections appears to be higher in boiler cleaning personnel.

Proceedings of Royal Society of Medicine, London

38:473-544 (July) 1945. **Partial Index**

Diphtheria Antigens: Their Preparation, Properties, Laboratory Testing and Statutory Control. P. Hartley.—p. 473.
*The Patient: Neglected Factor in History of Medicine. D. Guthrie.—p. 490.
The Paget Cell: Its Structure, Occurrence and Significance. E. Meirowsky and S. Keys, with technical assistance of P. H. Jacobs and Hyla M. Holden.—p. 495.
Treatment of Retained Testis. T. W. Mimpriss.—p. 507.

The Patient: Neglected in Medical History.—Guthrie pays tribute to some patients of the past who have played an important part in medical progress. The patient's name is mentioned in many of the descriptions of wonderful cures in the Greek temples of Esculapius. Hippocrates, though entirely free from this boastfulness, mentions the names of many patients. The contribution of the patient is passive, and few patients, even if they happen to be also medical men, are concerned with the benefit to humanity which may accrue from their sufferings. The author mentions Beaumont's patient Alexis St. Martin and Mrs. Jane Todd Crawford, on whom Dr. Ephraim McDowell performed the first ovariotomy. Louis Pasteur gave full credit to his patients for the part they played in the fight against rabies. The story of the Alsatian boy Joseph Meister, who was attacked by a mad dog, is well known. On May 14, 1796 Jenner inoculated James Phipps, a boy 8 years of age, with cowpox from the hand of Sarah Nealmes, a milkmaid. There have always been doctors who did not hesitate to experiment on themselves in the interests of science; men like John Hunter, who shortened his life by an unfortunate effort to investigate the cause of syphilis. Lazear, Adrian Stokes and Noguchi died of yellow fever, Ricketts of typhus, Dutton of trypanosomiasis, and there have been many other martyrs to science. Banting related that the first patient to be treated with insulin, in 1922, was Dr. Joe Gilchrist. In the category of self experiment may be placed Manuel Garcia, the singing master who was curious to see his own vocal cords and who invented the laryngoscope in 1854, although he had no idea then that he was founding a new medical specialty. Among the patients whose influence has been of a negative character Guthrie mentions "typhoid Mary."

Archives des Maladies de L'Appareil Digestif, Paris

34:1-100 (Jan.-Feb.) 1945. **Partial Index**

Digestion of Cellulose in Man. M. Chiray, J. Pochon and J. Dierick.—p. 1.
Invagination in Adults Induced by Intestinal Tumors. Bergeret, Mégret and Champeau.—p. 14.
*Nonulcerative Duodenitis. P. Oury and A. Vernet.—p. 30.
Bacteriologic Contribution to Problem of Abdominal Gas: Limitations of Knowledge. A. R. Prévot.—p. 45.
Ulcer of Rectum. Monique Parturier-Lannegrace and J. Arnous.—p. 50.
Disturbances of Digestion Secondary to Excess of Estrogens. M. A. Bernard.—p. 56.

Nonulcerative Duodenitis.—Duodenitis is to be differentiated from ulcer of the duodenum as gastritis is differentiated from gastric ulcer. Dyspeptic, pseudoulcerative and hemorrhagic duodenitis can be diagnosed by clinical features, but the diagnosis of duodenitis is difficult. Serial roentgenograms may clarify the diagnosis and may aid in the differential diagnosis from ulcer. Oury and Vernet do not agree with many radiologists and clinicians that any deformity of the bulb is a definite sign of ulcer. The authors emphasize that only a niche on the lesser or greater curvature is characteristic of an ulcer. Rarely the niche may be visualized at the base. In the absence of a niche, additional important signs of ulcer are an incisura caused by spasm, pseudodiverticula from a juxtapyloric ulcer, a true bulbar biloculation and a deformity of the bulb. In contrast to this, duodenitis presents a hazy bulbar contour in the absence of a niche, rapid variations in the configuration of the deformity and difficulty in filling and emptying of the bulb, with irregular indented borders. Medical treatment is to be preferred to surgical treatment. The treatment consists in a strict prolonged diet of milk, vegetable soups, semolina and tapioca soups, to which are added toasted bread, stewed fruit, lean meat and lean fish. Rest is helpful. Administration of 5 Gm. of bismuth subnitrate before meals and tincture of belladonna, 30 drops as the total daily dose, is recommended. In belladonna resistant cases good results were obtained by intravenous injections of atropine. Subcutaneous administration of solution of parathyroid combined with intravenous injections of calcium gluconate was effective in cases of pseudoulcerative duodenitis. Administration of vitamin A and D is favored.

Cardiologia, Basel

9:1-120 (Nos. 1 & 2) 1945

Clinical and Anatomic Investigations on Patient with Winiwarter Buerger's Endangiitis Obliterans Who Had Been Observed for Twenty-Four Years. V. F. Wuhrmann and A. Essellier.—p. 1.
Electrocardiographic Diagnosis of Location of Branch Block: Critical Analysis and Casuistic Study; Clinical, Electrophonocardiographic, Roentgenokymographic and Sphygmographic Study; Histologic Study. F. Addarii, L. Martini, I. Mahaim and M. R. Winston.—p. 33.
*Examination of Capillary Resistance in Human Subjects. V. W. Frischknecht.—p. 76.
Principles of Cardiovectography: II. Stereography. R. Sulzer and P. W. Duchosal.—p. 106.

Capillary Resistance in Human Subjects.—Frischknecht found that the suction procedure (principle of Hecht, 1907) is superior to the pressure method (principle of Rumpel-Leede, 1911) in determining capillary resistance. He determined the critical pressure values in 15 cases with a 2 cm. suction bell and compared these values with results obtained with a 1 cm. suction bell. The measurements were made in three regions: supraclavicular, infraclavicular and cubital. It was found that a person can show pathologic values in the clavicular regions and normal values in the cubital region or vice versa. A constant relation between the critical pressure obtained with the 2 cm. or 1 cm. bell was not observed. The critical time is not a suitable measure, but the critical pressure is the optimal measure for capillary resistance in that fluctuations of the clinical pressure parallel the clinical course of hemorrhagic diathesis. A single determination of capillary resistance obtained with the suction method frequently gives less information than the stasis experiment of Rumpel-Leede, since the normal values of the critical pressure vary greatly. The aim of the suction method is not the determination of the absolute capillary resistance but control of the fluctuations of the resistance during the action of various stimuli. This can be ascertained by daily determination in the supraclavicular region of the critical pressure, which is that negative pressure (measured in millimeters of mercury) which produces one to two petechiae in one minute suction time under a bell 2 cm. in diameter.

Acta Chirurgica Scandinavica, Stockhlom

92:1-98 (March 10) 1945

*Ten Years of Serum Therapy of Appendicitis. C. D. Bartels and E. Manicus-Hansen.—p. 1.
Rectal Prolapses in Children. O. Wiklander.—p. 17.
Obstruction Following Gastric Resection and Gastroenterostomy. R. Brandberg.—p. 37.
Study of Purpuroid Diseases of Urinary Tract. M. Sulamaa.—p. 65.
Stabilizing Operation for Asymmetrical Sacralization. K. E. Petersen.—p. 86.

Serum Therapy of Appendicitis.—Bartels and Manicus-Hansen compared the results of operations for acute appendicitis during the decade from 1931 to 1940, in which serum therapy was practiced, with those of 1921 to 1930 without serum therapy. During the period without serum therapy 490 operations were performed with 48 deaths, as compared with 902 operations with 28 deaths during the period in which serum therapy was employed. The number of cases of peritonitis was nearly the same within the two periods. Accordingly, differences in the severity of the cases cannot possibly have played any part in the decrease of the mortality rate within the period during which serum therapy was employed. Adults generally received 20 to 25 cc. of anti-gas gangrene serum and 25 cc. of anti-coli serum. Children received correspondingly smaller doses. Intravenous administration of serum was performed at the end of the operation. Larger doses were given in severe cases. The injections were repeated during the first few days after the operation. The serum is not only a "peritonitis serum." It should also be given as a prophylactic measure in gangrenous cases and in cases in which the disease is of more than forty-eight hours' duration.

Book Notices

Bacillary Dysentery, Colitis and Enteritis. By Joseph Felsen, B.A., M.D., Director of Medical Research, Bronx Hospital, New York. Cloth. Price, $6. Pp. 618, with 144 illustrations. Philadelphia & London: W. B. Saunders Company, 1945.

For many years in the temperate climes bacillary dysentery was regarded as being either nonexistent as a clinical entity or characterized only by occasional sporadic endemics. That *Public Health Reports* constantly reported, month by month, the recurrence of occasional cases of dysentery seemed of no moment or consequence and was generally ignored. The author of this book has been one of the foremost proponents of the thesis that carriers of this dreaded disease were always among us, that endemics were of frequent occurrence and that the chronic form of bacillary dysentery was constantly to be observed. The further theory that dysentery was in a direct manner associated with ulcerative colitis and even enteritis was his particular thesis, one which in past years he has labored hard to establish. By the collaborated studies of the International and the Pan American Dysentery Register he has had an unusual opportunity to collate all this material in a well written, readable, interesting and well documented volume.

The prose style of the book is excellent; the references are innumerable, accurate and extremely up to date. The typography, the paper, the illustrations, are excellent and well reproduced.

The historical aspects of bacillary dysentery are well analyzed; the momentous effects of such epidemics in terminating military campaigns, rather than the force of arms, are startlingly portrayed and are only now to be appreciated after the recent armed struggle, in which for the first time in history epidemics of disease were notably absent.

The epidemiology and the clinical aspects of epidemic and endemic bacillary dysentery are described with true clinical instinct, and the underlying bacteriology and serology, confusing as it is with its various and varying classes and nomenclature, are depicted in relative simplicity. The book, in this respect, fulfils a long-needed want for the general reader.

By including chronic ulcerative colitis and chronic distal ileitis as subtitles of bacillary dysentery, the author begs his question and presupposes the etiologic relationship of the two or more clinical entities. The latter part of the book is an attempt to define and to defend this hypothesis. Some confusion arises when in the statistical treatment of etiology the author constantly refers to "diarrhea, enteritis and dysentery" as if they were all one. Statements that chronic dysentery can be differentiated from ulcerative colitis by means of the sigmoidoscope run counter to the oft expressed opinions of many of our foremost authorities. The absolute clinical significance which the author grants to low titers of serum agglutination is also much mooted and less acceptable to many bacteriologists and clinicians. The fact that diagnostic titers were obtained in 93.5 per cent of cases of ileitis and colitis is a striking statement.

Regardless of whether one agrees in whole or in part with the thesis of the author, the book contains an orderly presentation of all the facts to date, clearly and lucidly laid out, arranged with precision and scientific discrimination, a volume which is both literary and provocative and essential reading for internists and specialists, who will see this problem grow in importance and magnitude, rather than recede, in coming years.

Bacillary dysentery is unquestionably among us at all times, and the relationship of the disease to the growing problems of colitis and ileitis deserve the very close study which this volume has devoted to the problem.

Introduction to Diseases of the Chest. By James Maxwell, M.D., F.R.C.P., Assistant Physician and Demonstrator of Practical Medicine, St. Bartholomew's Hospital, London. Second edition. Cloth. Price, 12s. 6d. Pp. 292, with 66 illustrations. London: Hodder and Stoughton, Ltd., 1945.

This edition of Dr. Maxwell's book has been brought up to date in several respects, yet it is shorter and contains fewer illustrations than the first, which was reviewed in these columns in 1939.

The book is divided into four sections and thirty-six chapters followed by the illustrations, which are individually captioned and referred to in the text. Sections I, II and III deal with the fundamentals of diagnosis of chest diseases. The history is concisely developed by discussion of major symptoms, minor symptoms and auxiliary data. Emphasis is placed on the major symptoms of chest disease in this first section and they are often referred to in subsequent chapters.

Dr. Maxwell has done an admirable piece of work in his presentation of the principles of physical examination of the chest. Emphasis has been placed on careful inspection of the entire patient and then inspection of the chest before any further examination is made. In a concise and clear manner the technic of palpation, percussion and auscultation is described. Similarly the significance of the physical findings is described in a manner making this section valuable particularly to the student and a refresher for the practitioner.

Attention should be drawn to the summaries concluding the chapters on physical examination. Likewise section III, on special investigations, clearly describes sputum studies, the value of hematologic studies and examination of the pleural fluid. Chapter ii, on radiology of the chest, is well written and contains a wealth of information. It is good to see emphasis given to the place held by thoracoscopy and exploratory thoracotomy in chest diagnosis.

Chapters xiii to xxxvi in section IV deal with diseases and conditions of various parts of the respiratory tract. It is enlightening to find a work on chest diseases in which diseases of the upper respiratory tract and larynx precede any discussion of bronchopulmonary disease. Reference is made in the chapter on bronchiectasis to the influence of upper respiratory infection on bronchopulmonary function and disease.

The chapter dealing with asthma is well written and develops several factors any one of which may be the stimulus setting off the asthmatic syndrome. These factors are (1) allergic, (2) nasal, (3) bronchopulmonary, (4) alimentary, (5) endocrine and (6) psychologic. Their interrelation is stressed. Other chapters dealing with chest injuries, pulmonary circulation, emphysema, lung abscess, pleurisy, empyema, pulmonary tuberculosis and tumors of the bronchi and lungs are clear descriptions of these entities, their diagnosis and treatment.

More elaborate information on and discussion of silicosis, other industrial pulmonary diseases and some of the less common chronic lung infections would be welcome. This would, of course, enlarge the text.

Dr. Maxwell's consideration of the treatment of pulmonary tuberculosis is sound and conservative, and a brief discussion of surgical procedures is presented.

This small volume contains a wealth of material. The author has wasted no words but rather has presented his subject so directly and concisely that both the student, the general practitioner and the specialist may all find here the meat of an important subject.

The Care of the Neurosurgical Patient Before, During and After Operation. By Ernest Sachs, A.B., M.D., Professor of Clinical Neurological Surgery, Washington University School of Medicine, Saint Louis. Cloth. Price, $6. Pp. 268, with 177 illustrations. St. Louis: C. V. Mosby Company, 1945.

This little book will be a source of pleasure to every one interested in neurologic surgery. It was written by one of the pioneers and masters of this field of surgery. As the title indicates, it is primarily concerned with the actual care of the patients, although the questions of symptoms, signs and diagnosis are touched on. The major concern is with the preoperative preparation of the patient, the organization of the operating room, the choice and administration of the anesthetic, actual operative technics, the administration of fluids and the postoperative care of the patient. The book is intensely personal. Dr. Sachs has given us the benefit of his many years of experience. He has related in detail how his patients are cared for at Barnes Hospital. The book will prove a mine of information, particularly to young men just beginning their training in neurologic surgery. It is replete with details of those little things which mean so much in the satisfactory handling of a difficult situation and in achieving all of the comfort possible for the patient.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

RH FACTOR, ABORTIONS AND STERILITY

To the Editor:—I have been married for ten years and have no children. My wife has had three miscarriages. She is Rh negative. For the past four years she has been unable to conceive. Could the Rh factor be responsible for this as well as for the miscarriages?

Captain, M. C., A. U. S.

ANSWER.—The extensive data thus far accumulated have failed to show any etiologic connection between the Rh factor and repeated miscarriages or sterility. In any event, as many as 1 out of every 7 persons is Rh negative, so there is a strong possibility of coincidence in a case like this. Clinical complications occur only when those who are Rh negative become sensitized to the Rh factor (only 1 in about 30 becomes sensitized when exposed to Rh positive blood by transfusion or as a result of a pregnancy with an Rh positive fetus), and the state of being Rh negative is no cause for concern as long as sensitivity is absent. Reliable tests for Rh antibodies are now available which can be carried out on any patient's blood serum and will definitely establish whether or not one is sensitized to the Rh factor (Wiener, A. S.: Conglutination Test for Rh Sensitization, *J. Lab. & Clin. Med.* **30**:662 [Aug.] 1945. Diamond, L. K., and Abelson, N. M.: The Detection of Rh Sensitization: Evaluation of Tests for Rh Antibodies, *ibid.* **30**:668 [Aug.] 1945. Coombs, R. R. R.; Mourant, A. E., and Race, R. R.: Detection of Weak and "Incomplete" Rh Agglutinins: A New Test [Preliminary Report], *Lancet* **2**:15 [July 7] 1945).

SUFFOCATION AND BLOOD CARBON MONOXIDE

To the Editor:—It was stated recently in connection with a death presumably caused by suffocation that, since no carbon monoxide was found in the blood fifteen days after death, the death could not have resulted from suffocation, as in suffocation there would be incomplete combustion of the tissue, and carbon monoxide would be found in the blood. In suffocation, does not death result from absence of combustion rather than from incomplete combustion? Even if any carbon monoxide was formed, would it not be found as carbon monoxide hemoglobin rather than as carbon monoxide gas? There were not any postmortem signs of carbon monoxide poisoning reported by the pathologist. Could carbon monoxide be found in blood fifteen days after death? Would not a negative test for carbon monoxide in blood be without significance in death by suffocation? How long after death could carbon monoxide hemoglobin be detected?

Francis L. Parker, M.D., Charleston, S. C.

ANSWER.—W. D. McNally of Chicago in his book on Toxicology, 1937, cites a case in which he found carbon monoxide in the tissues of a body examined one year after death. The literature on the subject is reviewed in C. K. Drinker's book on Carbon Monoxide Asphyxia, 1938. There a case is cited in which carbon monoxide was found two hundred and ten days after death. Its presence or absence in the exhumed body depends on several factors. For example, even in a patient resuscitated from near death by artificial respiration and carbogen (carbon dioxide 7 per cent and oxygen 93 per cent) the blood is not completely cleared of carbon monoxide for from twelve to twenty-four hours. This is because carbon monoxide diffuses into the tissues and is fixed by respiratory pigment in the liver, spleen, bone marrow and muscle, which releases the gas rather slowly. After death from carbon monoxide poisoning, diffusion to the surrounding air would be slow and would reach an equilibrium in an airtight coffin; this would not occur in the presence of air currents. If the body decays and is leached with water, presence or absence of carbon monoxide would depend on the extent of the decay and leaching. Thus the interpretation of a negative test for carbon monoxide in the blood or tissues of a body would depend on the conditions of the burial and the state of the body on being exhumed.

It is likely that, unless the blood vessels of a corpse have been perfused considerably before the embalming fluid is injected, the tissues will show the presence of carbon monoxide much longer than fifteen days. A thorough perfusing of the blood vessels of a person dying of carbon monoxide poisoning would remove most of the blood and some of the carbon monoxide from the tissues. The blood vessels then could be injected with embalming fluid and no carbon monoxide would be found in the contents of the large blood vessels. In a case of this sort the so-called blood could be negative for carbon monoxide and the tissue content would be small. However, if the person had died of carbon monoxide poisoning and the corpse was not tampered with, then carbon monoxide should be present fifteen days after death in any blood found and in the tissues in significant amounts. The finding of a positive test of low concentration is not easy to interpret. Smoking may yield a carbon monoxide saturation of the blood of from 1 to 5 per cent. Also the embalming fluid may be saturated with carbon monoxide gas and cause a positive test, or blood may be removed from a donor, saturated with carbon monoxide and then injected into the corpse.

REFRACTORY MENOPAUSAL SYMPTOMS

To the Editor:—A woman aged 42, following a complete hysterectomy, developed surgical menopause symptoms while in the hospital. She has been receiving 40,000 to 60,000 rat units of estrogenic substances hypodermically and six proprietary estrogenic tablets daily. Despite this she still is uncomfortable and has menopausal symptoms. How high can I go safely with the estrogens hypodermically? Would there be any benefit if I were to give less estrogen and some lutein extract? No benefit has been noted from ovarian and pituitary extract tablets. I have been using both the oil ampules and the aqueous ampules, but the patient still complains of pain, and swellings develop at the site of injection even though the area is massaged.

M.D., New York.

ANSWER.—Apparently this patient is suffering from a severe menopausal syndrome which is extremely resistant to estrogenic therapy, since relatively large amounts of estrogens have been found ineffective in relieving the symptoms. Occasionally such patients are encountered, and it is usually found that there is an underlying psychic pattern which offers resistance to any form of medication. It is possible that the patient has a psychic disturbance which may require treatment apart from the endocrine therapy. That she may be an extremely high strung and suggestible individual is indicated by the fact that she reacts locally to the aqueous suspensions of estrogens which are without any chemical irritant.

The following treatment is recommended to determine whether or not the patient is simply resistant to estrogen therapy or has an underlying psychic disturbance: She should receive injections of 5 mg. of estradiol dipropionate or diethylstilbestrol dipalmitate once weekly for three weeks. If this does not produce a satisfactory therapeutic response there is no need to continue searching for the proper dosages of estrogens, since this dosage is ample for all such cases. The continuous powerful action of these two substances makes them the most effective estrogens available. If this therapy is not effective, psychiatric consultation is advised. If this is not feasible, sedation with phenobarbital, ¼ or ½ grain (0.016 or 0.032 Gm.) three times daily, may be used in conjunction with estrogenic therapy. One other possibility remains: combination of the large dose of estrogens with 10 mg. of testosterone propionate at each injection, since occasionally it has been found that this therapy may work to advantage.

IRRITATION FROM USE OF CONTACT LENSES

To the Editor:—Will you kindly send information concerning the use of contact lenses by a patient with keratoconus? I am referring to such problems as methods of getting rid of excess mucus and causes of "steam" under the lens.

A. V. Carl, M.D., Danville, Pa.

ANSWER.—These difficulties are all due to irritation, which may be mechanical or chemical. Those due to mechanical irritation can be helped by a better fit to remedy pressure points or tight or loose fitting and thick edges. There is a tendency to increased tolerance as the contact glass is worn, so that one must not be discouraged by an initial reaction that comes on in half an hour to an hour during the first week or two of use. Fortunately, cases of keratoconus are among the more tolerant, less sensitive ones.

More difficult is the treatment of the chemical irritation. The lubricating solution may be of too low, more rarely too high, osmotic strength. Usually a solution isotonic with the tears equivalent to 1.4 per cent sodium chloride is best. The solution may not be sufficiently alkaline. Usually a p_H of 8 to 8.8 is well tolerated. The clouding may be due (*a*) to the presence of mucus or other secretions and cells which find their way into the liquid lens between the cornea and the contact glasses or (*b*) to opacities forming in the cornea itself. The former disappear at once with a fresh clean solution. The latter disappear slowly in fifteen minutes or more, depending on how far they have progressed. Different buffer solutions must be tried, the concentration and the p_H being varied and different chemicals being used, since there are idiosyncrasies—for example, some persons do not tolerate boric acid. If clouding occurs

after a definite period, say two or four hours, the patient should be instructed to change the solution about half an hour before the clouding usually takes place.

Formulas for buffer solutions:

A. Solution 1 (acid)

Boric acid	12.4 Gm.
Sodium chloride	8.4 Gm.
Distilled water	1,000.0 cc.

Solution 2 (alkaline)

Sodium borate anhydrous (equivalent to 36.2 Gm. of borax)	19.1 Gm.
Sodium chloride	11.7 Gm.
Distilled water	1,000.0 cc.

To obtain buffer of desired p_H, these are mixed as follows:

p_H	Solution 1	Solution 2
7.6	30	5.25
8.0	30	11.0
8.4	30	18.0
8.8	15	45.0

B. Solution 1

Boric acid (anhydrous)	12.4 Gm.
Potassium chloride (anhydrous)	7.4 Gm.
Distilled water	1,000.0 cc.

Solution 2

Sodium carbonate anhydrous	21.2 Gm.
Distilled water	1,000.0 cc.

To obtain buffer of desired p_H, these are mixed as follows:

p_H	Solution 1	Solution 2
7.6	30	1.5
8.0	30	2.5
8.4	30	4.0
8.8	30	6.5

CHRONIC INFECTIOUS MONONUCLEOSIS

To the Editor:—**A boy aged 6 years has a positive heterophile antibody test for infectious mononucleosis. All other tests of the serum have been negative. General supportive measures are employed such as a high protein diet, crude liver extract and vitamins. I am concerned now as to whether or not the crude liver is indicated; I believe it is on a supportive basis. Also I should like suggestions as to the best way to get the proteins into his system and as to whether or not there are any specific vitamins supposed to be of benefit in conditions of this sort? There are signs of chronic inflammation of the liver—a mild hepatitis. In times past I have sometimes used sodium thiosulfate as an alterative. Would this drug have any special indication here? Would iodine benefit this patient? I have been cautioned against these drugs in view of the condition of the liver. Would convalescent serum be practical or applicable? Would immunotransfusions in small amounts be recommended? This case is rather protracted—now going into the seventh month. The patient seems to be holding up well, but the blood picture stays rather constant.**

L. F. Valentine, M.D., North Platte, Neb.

ANSWER.—It is rather unusual for the abnormal blood picture and high heterophile antibody to persist for seven months in infectious mononucleosis. There is no specific therapy for this disease. Convalescent serum (Lassen, H. C. A., and Thomsen, S.: *Acta med. Scandinav.* **104**:498, 1940) has been reported to be beneficial in the early stages in bringing down the fever and producing symptomatic relief, but the serum is not readily obtainable. Blood transfusions are not indicated unless there is a significant anemia. Reports on the use of immunotransfusions were not found. The combination of a high protein diet, brewers' yeast powder, acceptable multivitamin preparations and injections of crude liver have been recommended for the treatment of hepatitis but would have no specific effect on infectious mononucleosis. The protein content of the diet can be raised by giving supplements of oral amino acid mixtures (Aminoids, Arlington Chemical Company, or Amigen, Mead Johnson & Co.). Evidence is lacking that sodium thiosulfate or iodine would be beneficial in a case of this type.

PREGNANCY TEST AND ADMINISTRATION OF ESTROGENS

To the Editor:—**What effect would the administration of diethylstilbestrol have on the Friedman test in a nonpregnant woman? Would large parenteral injections of theelin affect the accuracy of the Friedman test?**

M.D., Chicago.

ANSWER.—The administration of estrogenic substances to a patient should not affect the accuracy of the Friedman test if the determination is made on the basis of the ovarian reaction in the rabbit which receives the injection of the patient's urine. This reaction consists in the development of hemorrhagic follicles in the otherwise small nonvascular rabbit's ovaries. Some laboratories utilize the development of hyperemia of the oviducts in the rabbit as an indication of a positive test for pregnancy in the absence of ovarian development. This reaction depends on the response to estrogens from the rabbit's ovaries or from the presence of estrogens in the urine. Obviously a patient who receives an administration of estrogens and who thus excretes a certain amount of it may give a false test when it is determined by the response of the oviducts. Where the reaction depends on the ovarian response to the chorionic gonadotropins in the patient's urine there should be no alteration in the accuracy of the test when the patient has received theelin or other estrogens.

TREATMENT OF CRUSHED FINGER

To the Editor:—**A patient had the distal end of his finger severely crushed. The distal phalanx was avulsed at the interphalangeal joint, and except for a small amount of soft tissue the crushed portion was all but amputated. The crushed tissue was sutured back in place with hope that there might be sufficient blood supply. The finger was splinted by the finger nail, which had remained in place, and by a tongue depressor which was incorporated in the dressing. In treating this case I ordered the extremity kept cold with an ice cap, for which I was criticized. The reason for ordering the cold treatment was to lower the metabolism in the distal end until a sufficient blood supply could be established.**

Lieutenant (jg), (MC), U.S.N.R.

ANSWER.—The best results in cases such as that described are probably accomplished by a judicious combination of compression and rest. The surgeon who transplants a flap of skin and subcutaneous tissue is always concerned about its vitality. In this case the surgeon was dealing essentially with a flap with a meager blood supply. Such flaps, as Blair has pointed out, are more likely to die from congestion and failure of the return circulation than from lack of sufficient blood. The venous return is greatly handicapped, more so than the arterial inflow. Compression aids the return circulation and helps to prevent the congestion that leads to tissue necrosis.

There does not seem to be any logical reason for the use of ice in such a case. The surgeon who transfers a flap from one part to another would not employ cold applications, simply because cold arrests tissue metabolism and leads to devitalization of tissue. Arrest of tissue metabolism is simply the early stage of tissue necrosis.

TOXICITY OF PHOSPHORIC ACID IN INDUSTRY

To the Editor:—**Will you kindly furnish me with information concerning the toxicity of phosphoric acid as used in factories for rust prevention.**

R. G. Hickerson, M.D., Keithsburg, Ill.

ANSWER.—Phosphoric acid as industrially employed for fingerprint removal and rust prevention is not a likely source of injury to exposed workers. It is no more damaging to the skin than an equal concentration of other mineral acids. Phosphoric acid in dilute form is frequently used by oral administration therapy in such quantities as 10 cc. every hour for ten hours. Difficulties currently being experienced in industry from phosphoric acid mixtures used for antioxidation usually derive from the solvent of phosphoric acid, which in many instances is butyl cellosolve. This compound is ethylene glycol mono-n-butyl ether and is credited with being the most toxic of the compounds in its series. Early manifestations center about minor irritations of the eyes and respiratory tract, but in more severe instances there may be inflammation of the liver and kidneys associated with the hematuria. In animals the minimum lethal dose is about 0.5 cc. per kilogram of body weight. Its apparent toxicity is about five times as high as that of the better known ethylene glycol. Other substances suitable as solvents of phosphoric acid are likewise toxic in varying degrees.

CANCER AND MENTAL DISTURBANCES

To the Editor:—**What investigation has been made of late years along the line of the effect of mental disturbances and their relation to cancer? I have been in practice over forty years, and my attention has been called many times to the development of cancer following long periods of mental worry over which the sufferer had no control.**

Wordsworth M. Elliott, M.D., Durango, Colo.

ANSWER.—Medical literature does not appear to contain reports of close studies of the relation of mental disturbances to the causation of cancer. This is certainly true for late years. So far as known there are no reliable statistics on the relative frequency of cancer in sane and insane persons of the same ages and other comparable states. The reports of hospitals for the insane do not contain accounts of notably large numbers of cases of cancer in their populations. While there is no evidence at hand that "worry" of itself can induce cancer, it is well known that the fear of cancer, cancerphobia, may be associated with mental disturbance. Of course it is not to be denied that worry associated with more or less important disturbances of metabolism and health may appear to favor the development of cancer under certain conditions. The frequency in advancing years of cancer and of states of "mental worry" makes it obvious that the two conditions may occur coincidently without any causative relationship.

JOURNALS ABSTRACTED IN THE CURRENT MEDICAL LITERATURE DEPARTMENT, SEPTEMBER—DECEMBER 1945

Titles have been listed or abstracts made of important articles in the following journals in the Current Literature Department of THE JOURNAL during the past four months. Any of the journals, except those starred, will be lent by THE JOURNAL to subscribers in continental United States and Canada and to members of the American Medical Association for a period not exceeding three days. Three journals may be borrowed at a time. No journals are available prior to 1933. Requests for periodicals should be addressed to the Library of the American Medical Association and should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Thus most of these journals are accessible to the general practitioner.

Acta chirurgica Scandinavica. Stockholm.
Acta dermato-venereologica. Stockholm.
Acta medica orientalia, Jerusalem.
Acta medica scandinavica. Stockholm.
Acta orthopaedica Scandinavica, Copenhagen.
American Heart Journal. St Louis.
American Journal of Clinical Pathology. Baltimore.
American Journal of Digestive Diseases. Fort Wayne, Ind.
*American Journal of Diseases of Children. A. M. A., Chicago.
American Journal of Hygiene. Baltimore.
American Journal of the Medical Sciences. Philadelphia.
American Journal of Obstetrics and Gynecology. St. Louis.
American Journal of Ophthalmology. Cincinnati.
American Journal of Orthopsychiatry, New York.
American Journal of Pathology Ann Arbor, Mich.
American Journal of Physiology. Baltimore.
American Journal of Psychiatry. New York.
American Journal of Public Health. New York.
American Journal of Roentgenol. and Radium Therapy. Springfield. Ill.
American Journal of Surgery. New York.
American Journal of Syphilis, Gonor. and Venereal Diseases. St. Louis.
American Journal of Tropical Medicine. Baltimore.
American Review of Soviet Medicine. New York.
American Review of Tuberculosis. New York.
Anais brasileiros de ginecologia, Rio de Janeiro.
Anesthesiology. New York.
Annales de dermatologie et de syphiligraphie, Paris.
Annales d'oto-laryngologie, Paris.
Annales pædiatrici, Basel.
Annals of Allergy. Minneapolis
Annals of Internal Medicine. Lancaster, Pa.
Annals of Otology Rhinology and Laryngology. St. Louis.
Annals of the Rheumatic Diseases. London.
Annals of Surgery. Philadelphia.
*Archives of Dermatology and Syphilology. A. M. A., Chicago.
Archives of Disease in Childhood, London.
Archives francaises de pédiatrie, Paris
*Archives of Internal Medicine. A. M. A., Chicago.
Archives des maladies de l'appareil digestif, Paris.
Archives des maladies professionelles, Paris.
*Archives of Neurology and Psychiatry. A. M. A., Chicago.
*Archives of Ophthalmology. A M A., Chicago.
*Archives of Otoloryngology. A. M. A., Chicago.
*Archives of Pathology. A. M. A., Chicago.
Archives of Physical Medicine. Chicago.
*Archives of Surgery. A. M. A., Chicago.
Archivos argentinos de pediatría, Buenos Aires.
Archivos del Hospital de niños Roberto del Río, Santiago.
Arizona Medicine. Phoenix
Australian Journal of Experimental Biology & Medical Science. Adelaide.
Australian and New Zealand Journal Surgery. Sydney.
Bollettino della Societa italiana de medicina e igiene tropicale, Asmara.
Brain. London.
Brasil-medico. Rio de Janeiro.
British Heart Journal London.
British Journal of Dermatology and Syphilis. London.
British Journal of Experimental Pathology. London.
British Journal of Industrial Medicine. London.
British Journal of Ophthalmology. London.
British Journal of Radiology. London.
British Journal of Surgery. Bristol.
British Journal of Tuberculosis London.
British Journal of Urology. London.
British Medical Journal. London.
Bulletin of the Johns Hopkins Hospital. Baltimore.
Bulletin of the Los Angeles Neurological Society. Los Angeles.
Bulletin of the New York Academy of Medicine. New York.
Bulletin of the U. S. Army Medical Department. Washington, D. C.
California and Western Medicine. San Francisco.
Canadian Journal of Public Health. Toronto.
Canadian Medical Association Journal. Montreal.
Cancer Research. Baltimore.
Chinese Medical Journal. Washington, D. C.
Connecticut State Medical Journal. Hartford.
Delaware State Medical Journal. Wilmington.
Diseases of Chest. Chicago
Edinburgh Medical Journal.
Endocrinology. Springfield, Ill.
Epidemiological Information Bulletin. Washington, D. C.
Gastroenterology. Baltimore.
Giornale di medicina, Palermo.
Gynécologie et obstétrique, Paris.
Hawaii Medical Journal Honolulu.
Helvetica medica acta, Basel.
Hospital, Rio de Janeiro.
Illinois Medical Journal. Chicago.
Indian Journal of Medical Research, Calcutta.
Indian Medical Gazette. Calcutta.
Irish Journal of Medical Science, Dublin.
Journal of Allergy. St. Louis.
Journal of the Arkansas Medical Society. Fort Smith.
Journal of Aviation Medicine. St. Paul.
Journal of Bone and Joint Surgery. Boston.
Journal of Clinical Endocrinology. Springfield, Ill.
Journal of Clinical Investigation. Boston.
Journal of Endocrinology. London.
Journal of Experimental Medicine. New York.
Journal of the Florida Medical Association. Jacksonville.
Journal of Hygiene. London.
Journal of Immunology. Baltimore.
Journal of the Indiana State Medical Association. Indianapolis.
Journal of Industrial Hygiene and Toxicology. Baltimore.
Journal of Infectious Diseases. Chicago.
Journal of International College of Surgeons. Chicago.
Journal of Investigative Dermatology. Baltimore.
Journal of the Iowa State Medical Society. Des Moines.
Journal of the Kansas Medical Society. Topeka.
Journal of Laboratory and Clinical Medicine. St. Louis.
Journal-Lancet Minneapolis.
Journal of the Maine Medical Association. Portland.
Journal of the Medical Association of the State of Alabama. Montgomery.
Journal of the Medical Association of Georgia. Atlanta.
Journal of the Medical Society of New Jersey. Trenton.
Journal of Mental Science. London.
Journal of the Michigan State Medical Society. Lansing.
Journal of the Missouri State Medical Association. St. Louis.
Journal of the Mount Sinai Hospital. New York.
Journal of the National Cancer Institute. Washington, D. C.
Journal National Malaria Society. Tallahassee, Fla.
Journal of Nervous and Mental Disease. New York.
Journal of Neuropathology and Experimental Neurology. Baltimore.
Journal of Neurophysiology. Springfield, Ill.
Journal of Neurosurgery. Springfield, Ill.
Journal of Nutrition. Philadelphia.
Journal of Obstetrics and Gynaecology of British Empire. Manchester.
Journal of the Oklahoma State Medical Association. Oklahoma City.
Journal of Pathology and Bacteriology. Edinburgh.
Journal of Pediatrics. St. Louis.
Journal of Pharmacology and Experimental Therapeutics, Baltimore.
Journal of Physiology. Cambridge
Journal of Royal Naval Medical Service. London.
Journal of the South Carolina Medical Association. Florence.
Journal of the Tennessee State Medical Association. Nashville.
Journal of Thoracic Surgery. St. Louis.
Journal of Urology. Baltimore.
Kentucky Medical Journal. Bowling Green.
Khirurgiya, Moscow.
Lancet. London.
Medical Annals of the District of Columbia. Washington.
Medical Journal of Australia. Sydney.
Medicina, Buenos Aires.
Medicina, Mexico.
Military Surgeon. Washington, D. C.
Minnesota Medicine. St. Paul.
Monatsschrift für Geburtshilfe und Gynäkologie, Basel.
Nebraska State Medical Journal. Lincoln.
New England Journal of Medicine. Boston.
New Orleans Medical and Surgical Journal.
New York State Journal of Medicine. New York.
Nordisk medicin. Gothenburg.
North Carolina Medical Journal. Winston-Salem.
Northwest Medicine. Seattle.
Ohio State Medical Journal. Columbus.
Pennsylvania Medical Journal. Harrisburg.
Physiological Reviews. Baltimore.
Practitioner. London.
Prensa Médica Argentina. Buenos Aires.

*Cannot be lent.

Presse médicale, Paris.
Proceedings of Royal Society of Medicine. London.
Psychosomatic Medicine. Baltimore.
Public Health Reports Washington, D. C.
Puerto Rico J. Public Health & Tropical Medicine. San Juan.
Quarterly Journal of Medicine. Oxford.
Quarterly Journal of Studies on Alcohol. New Haven, Conn.
Radiology. Syracuse, N. Y.
Review of Gastroenterology. New York.
Revue d'immunologie, Paris.
Revista chilena de pediatría. Santiago.
Rhode Island Medical Journal. Providence.
Rocky Mountain Medical Journal. Denver.
Sang, Paris.
Semana Médica. Buenos Aires.
South African Journal of Medical Sciences. Johannesburg.
South African Medical Journal. Cape Town.
Southern Medical Journal. Birmingham, Ala.
Surgery. St. Louis.
Surgery, Gynecology and Obstetrics. Chicago.
Texas State Journal of Medicine. Fort Worth.
Tubercle. London.
Union Médicale du Canada. Montreal.
United States Naval Medical Bulletin. Washington, D. C.
Virginia Medical Monthly. Richmond.
*War Medicine. A. M. A., Chicago.
Western Journal of Surgery Obstetrics and Gynecology. Portland, Ore.
West Virginia Medical Journal. Charleston.
Wisconsin Medical Journal. Madison.
Yale Journal of Biology and Medicine. New Haven.

SUBJECT INDEX

This is an index to all the reading matter in THE JOURNAL. In the Current Medical Literature Department only the articles which have been abstracted are indexed.

The letters used to explain in which department the matter indexed appears are as follows: "BI," Bureau of Investigation; "E," Editorial; "C," Correspondence; "OS," Organization Section; "ab," abstracts; the star (*) indicates an original article in THE JOURNAL.

This is a subject index and one should, therefore, look for the subject word, with the following exceptions: "Book Notices," "Deaths," "Medicolegal Abstracts" and "Societies" are indexed under these titles at the end of the letters "B," "D," "M," and "S." State board examinations are entered under the general heading State Board Reports, and not under the names of the individual states. Matter pertaining to the Association is indexed under "American Medical Association." The name of the author, in brackets, follows the subject entry.

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Acad.—Academy	*Indust.—Industrial*
Am.—American	*Internat.—International*
A.—Association	*M.—Medical*
Coll.—College	*Med.—Medicine*
Commn.—Commission	*Nat.—National*
Comm.—Committee	*Pharm.—Pharmaceutical*
Conf.—Conference	*Phys.—Physicians*
Cong.—Congress	*Soc.—Society*
Dist.—District	*Surg.—Surgery*
Found.—Foundation	*Surgs.—Surgeons*
Hosp.—Hospital	*S.—Surgical*

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Subscription, $8.00 | Published Weekly at | *Single Copy, 25 Cents*
VOLUME 129, No. 18 | 535 North Dearborn Street, Chicago 10, Illinois | DECEMBER 29, 1945

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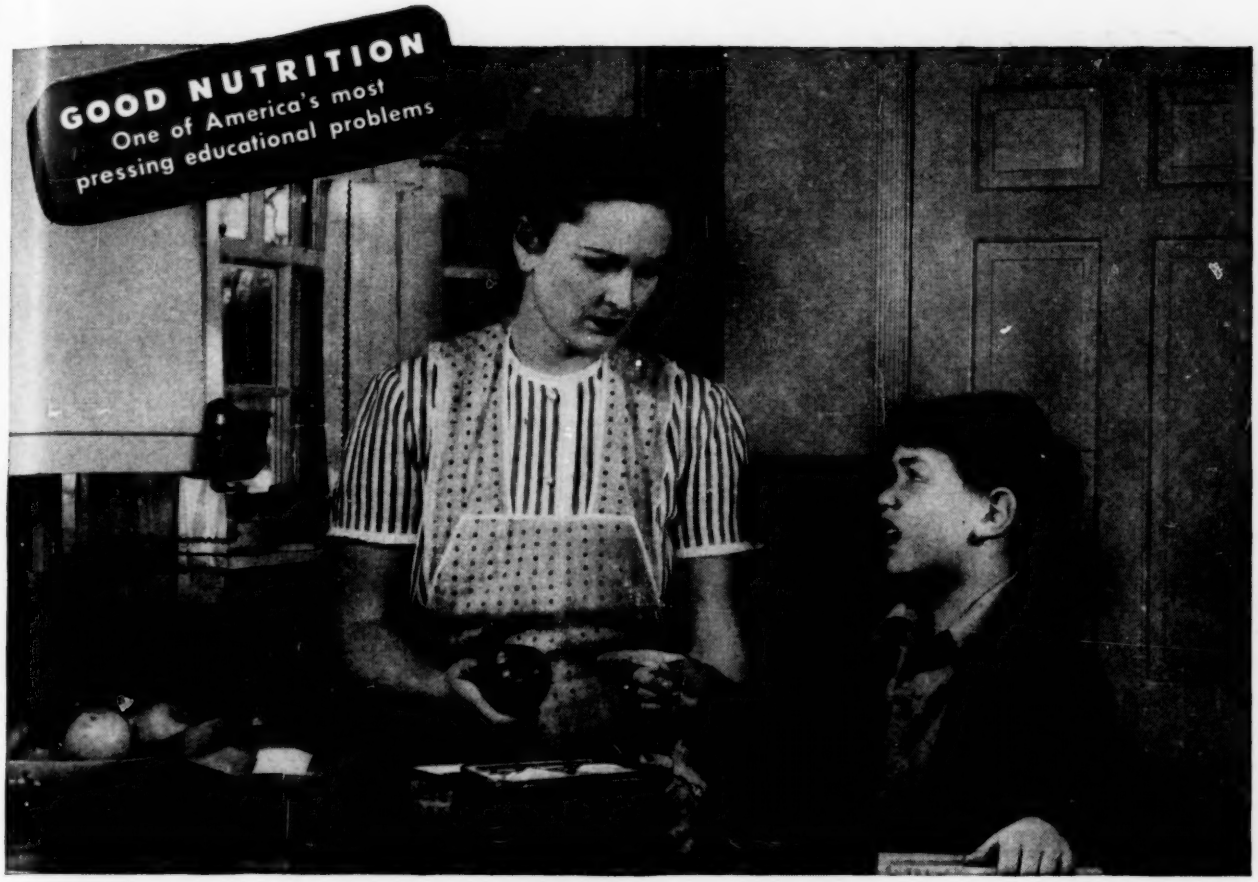

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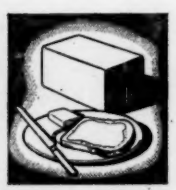

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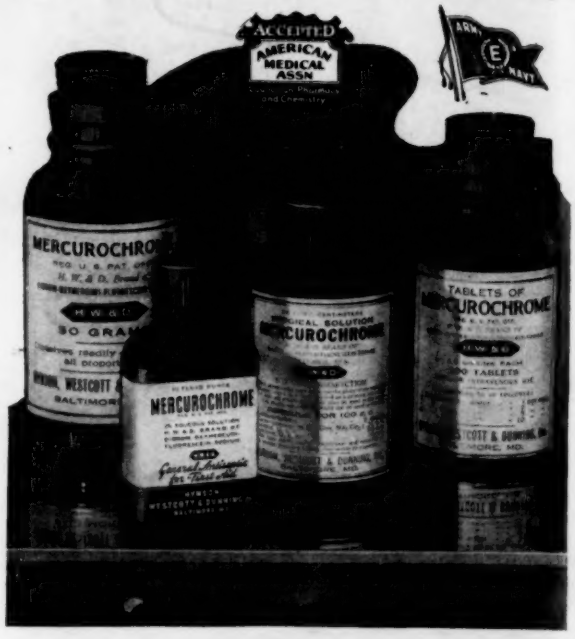

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FOR PHYSICIANS, SURGEONS, DENTISTS EXCLUSIVELY

ALL PREMIUMS COME FROM — PHYSICIANS SURGEONS DENTISTS — ALL CLAIMS GO TO

$5,000.00 accidental death $25.00 weekly indemnity, accident and sickness	**$8.00** Quarterly
$10,000.00 accidental death $50.00 weekly indemnity, accident and sickness	**$16.00** Quarterly
$15,000.00 accidental death $75.00 weekly indemnity, accident and sickness	**$24.00** Quarterly

ALSO HOSPITAL EXPENSE FOR MEMBERS WIVES AND CHILDREN

86¢ out of each $1.00 gross income used for members' benefit

$2,800,000.00 INVESTED ASSETS — **$13,000,000.00** PAID FOR CLAIMS

$200,000.00 deposited with State of Nebraska for protection of our members.

Disability need not be incurred in line of duty—benefits from the beginning day of disability

PHYSICIANS CASUALTY ASSOCIATION
PHYSICIANS HEALTH ASSOCIATION
43 years under the same management
400 FIRST NATIONAL BANK BUILDING • OMAHA 2, NEBRASKA